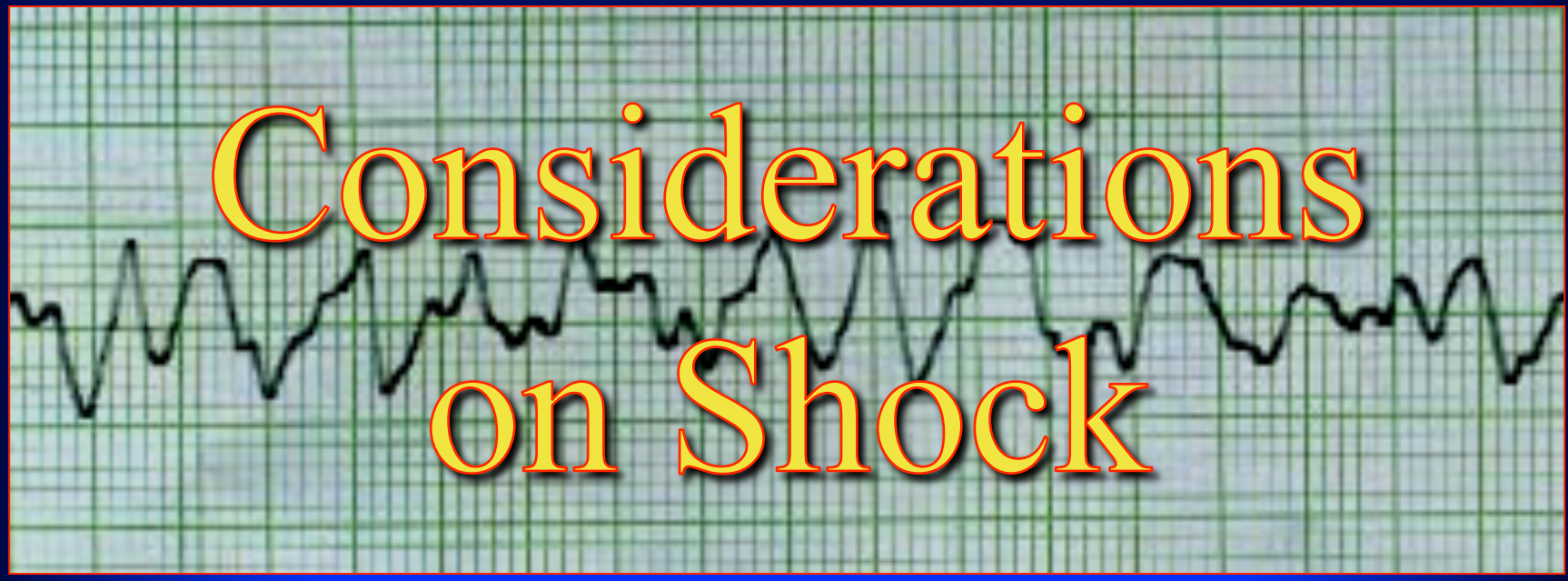

Considerations on Shock

Ruminations about the Past, Present, and Future

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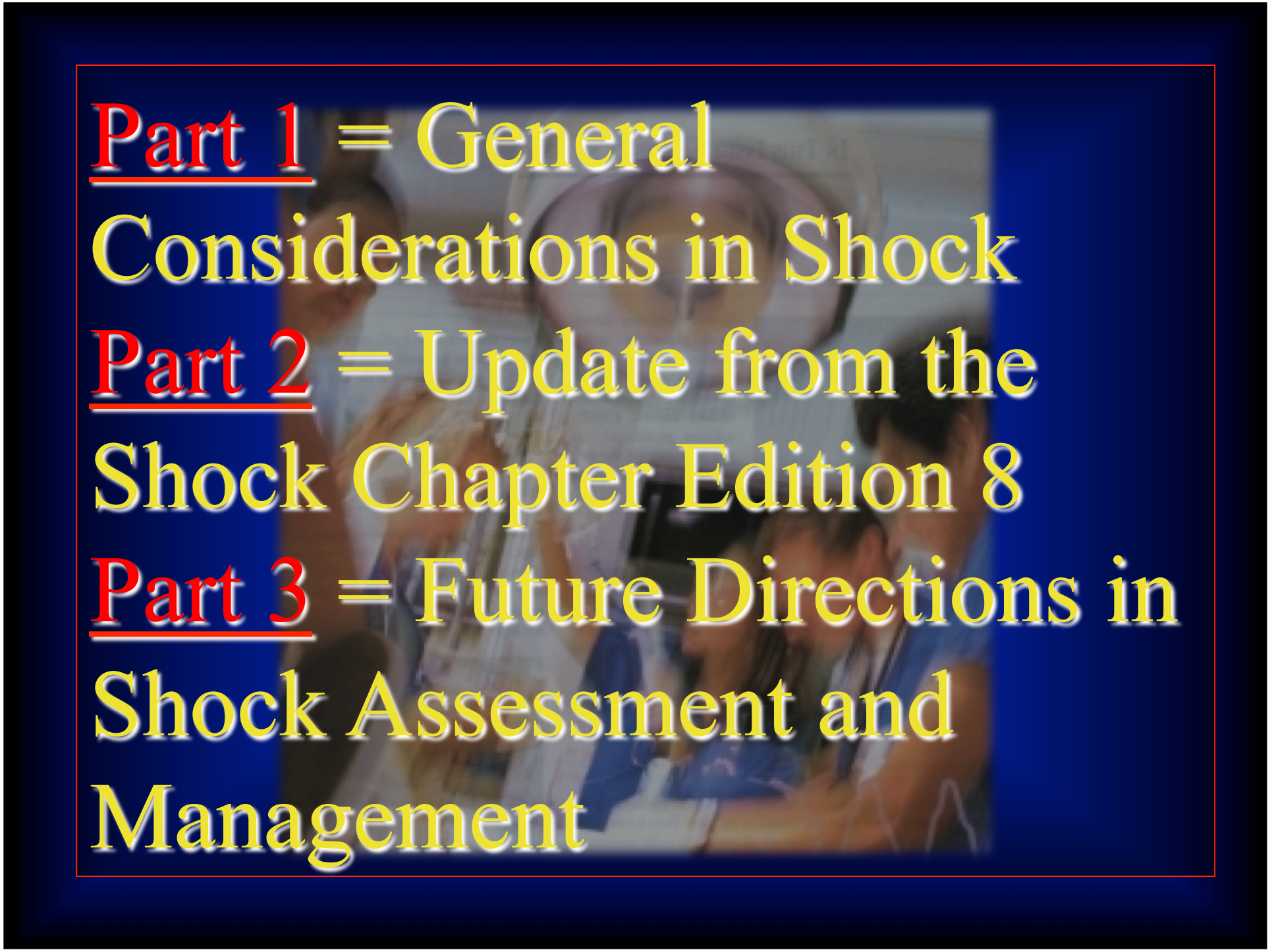
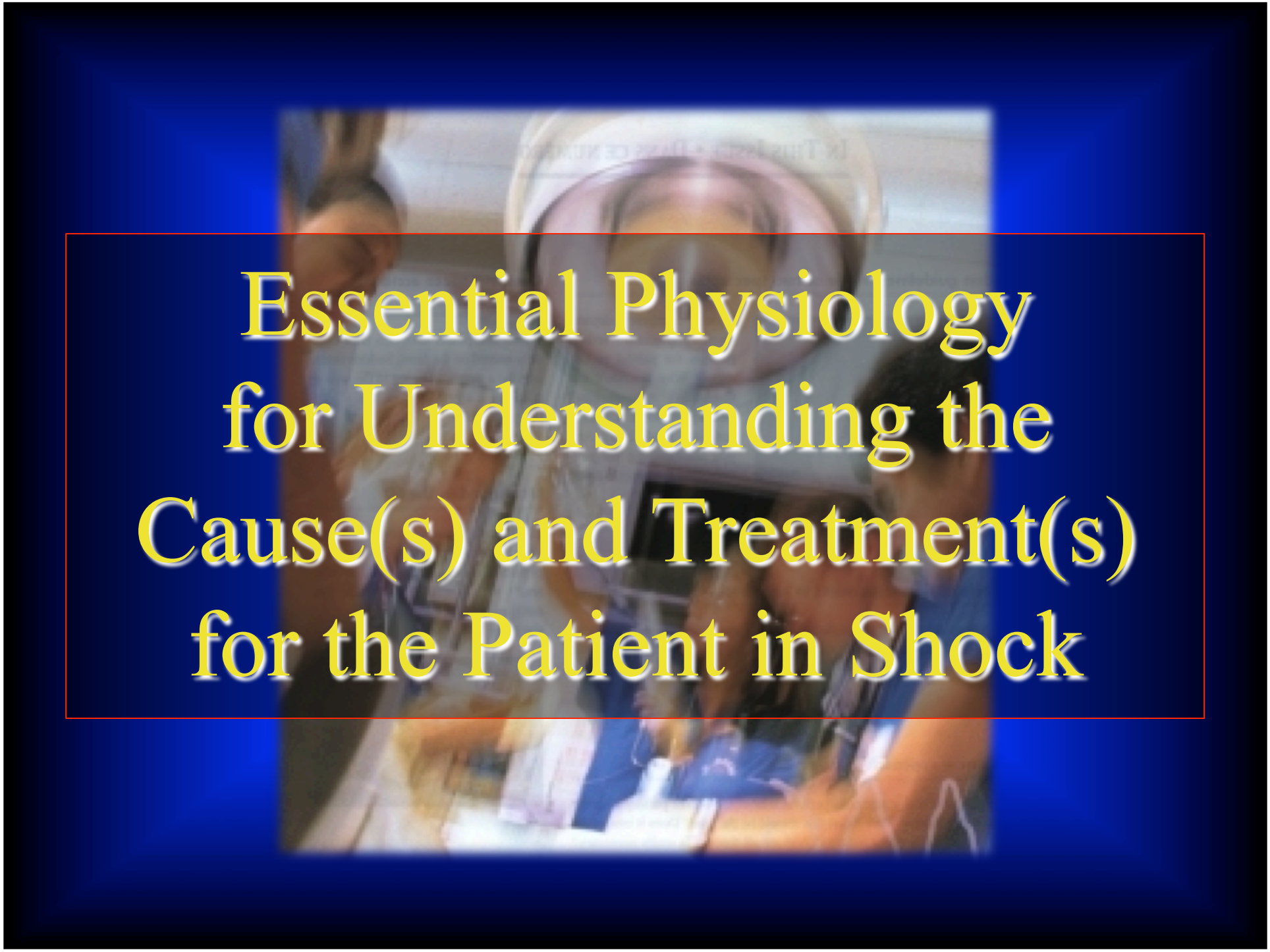
Division of Emergency Medical Services
Department of Emergency Medicine
UT Southwestern Medical Center


www.rayfowler.com

I have no disclosures to make.

I am full-time as academic faculty at UT Southwestern


Part 1 = General Considerations in Shock
Part 2 = Update from the Shock Chapter Edition 8
Part 3 = Future Directions in Shock Assessment and Management


Essential Physiology
for Understanding the
Cause(s) and Treatment(s)
for the Patient in Shock

Shock

"The term "*shock*" describes a condition that occurs when the perfusion of the body's tissues with oxygen, electrolytes, glucose, and fluid becomes inadequate to meet the body's needs."

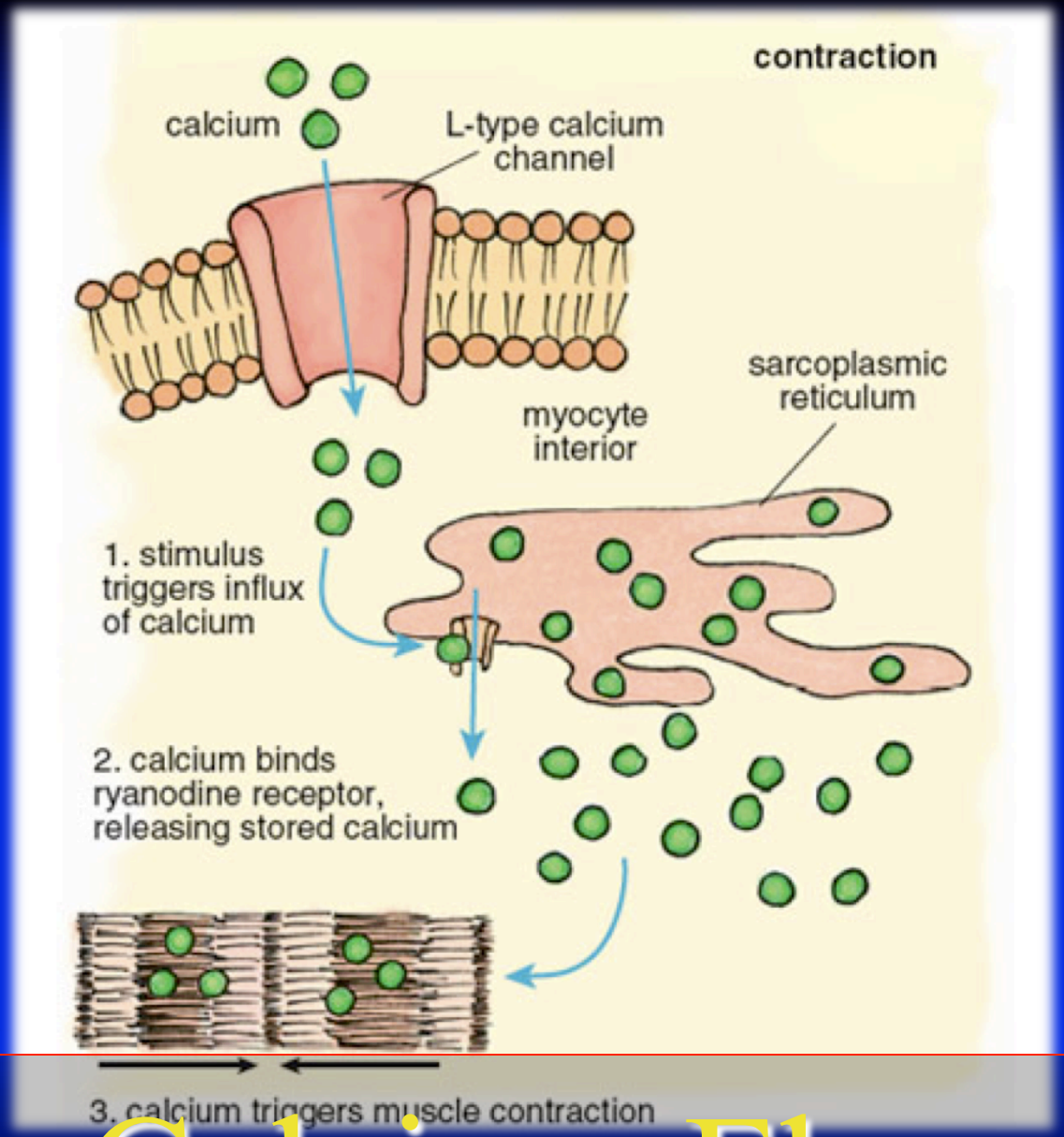
Shock

Deprived of oxygen, cells begin to use "backup" processes, which make energy for the body less efficiently and produce toxic by-products such as lactic acid.

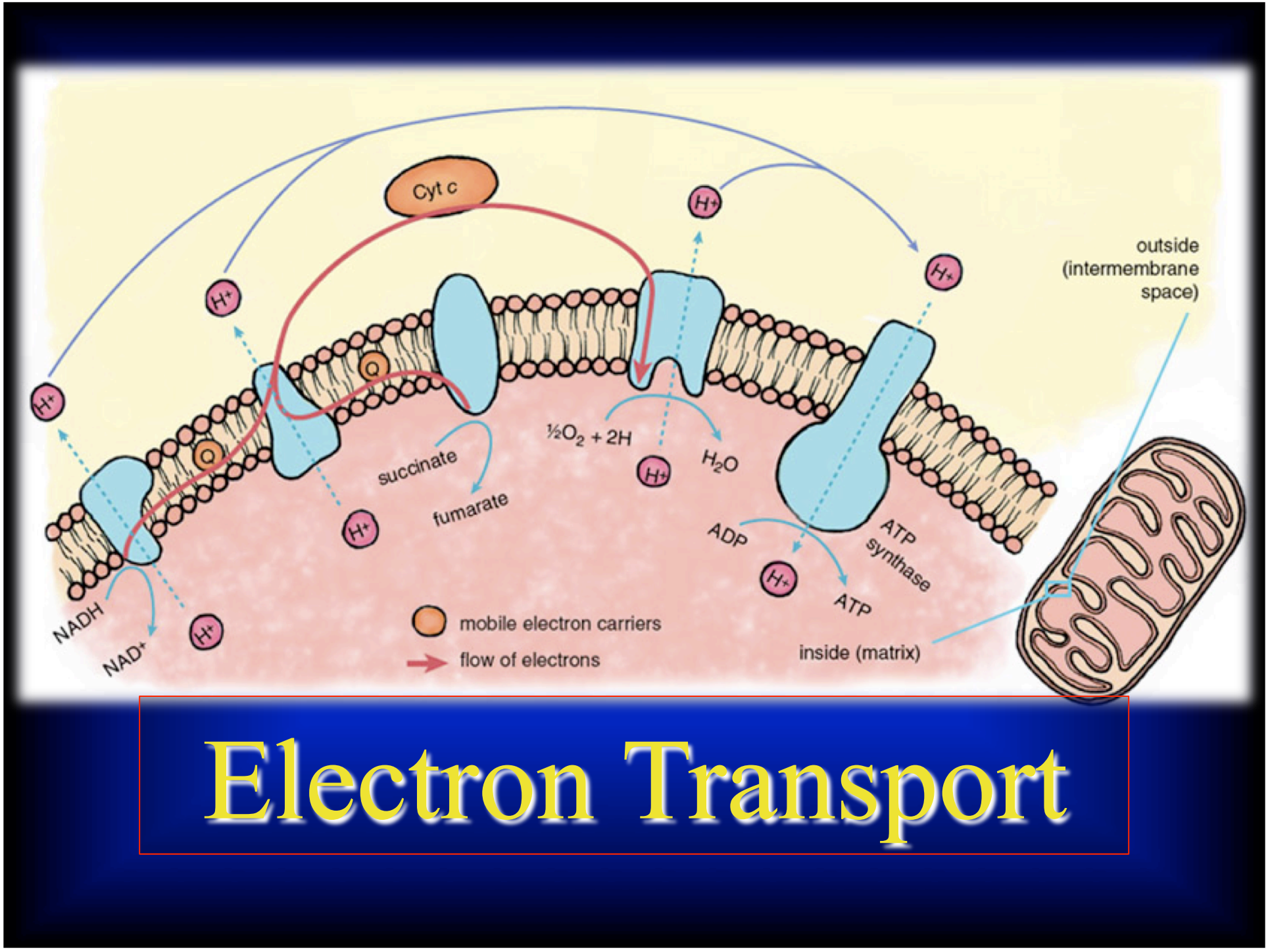
The backup (anaerobic) processes may postpone cellular death for a time.

Shock

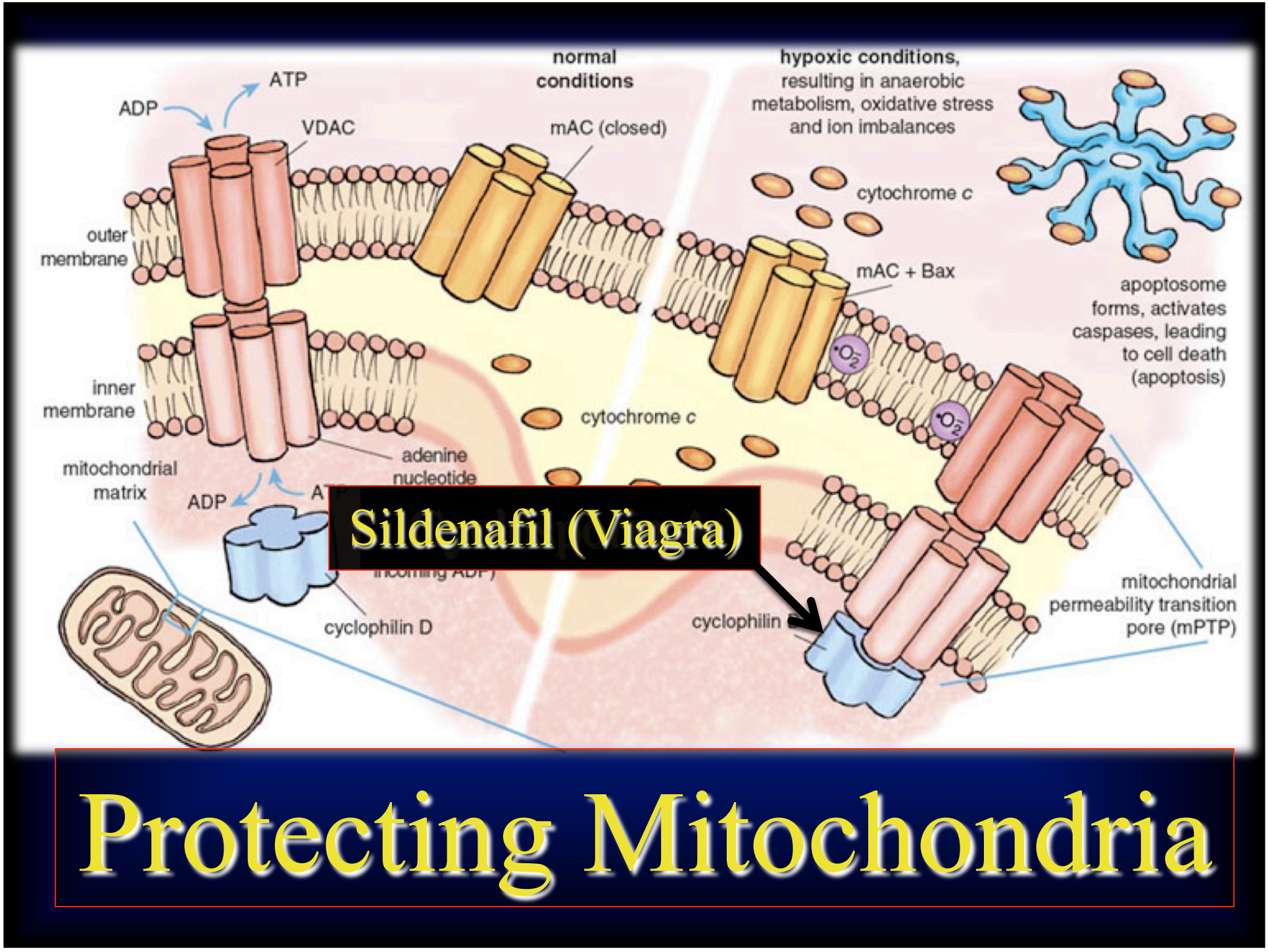
However, the lack of oxygen is compounded by those toxic by-products because they can poison certain cellular functions, such as the production of energy by mitochondria.



Calcium Flux



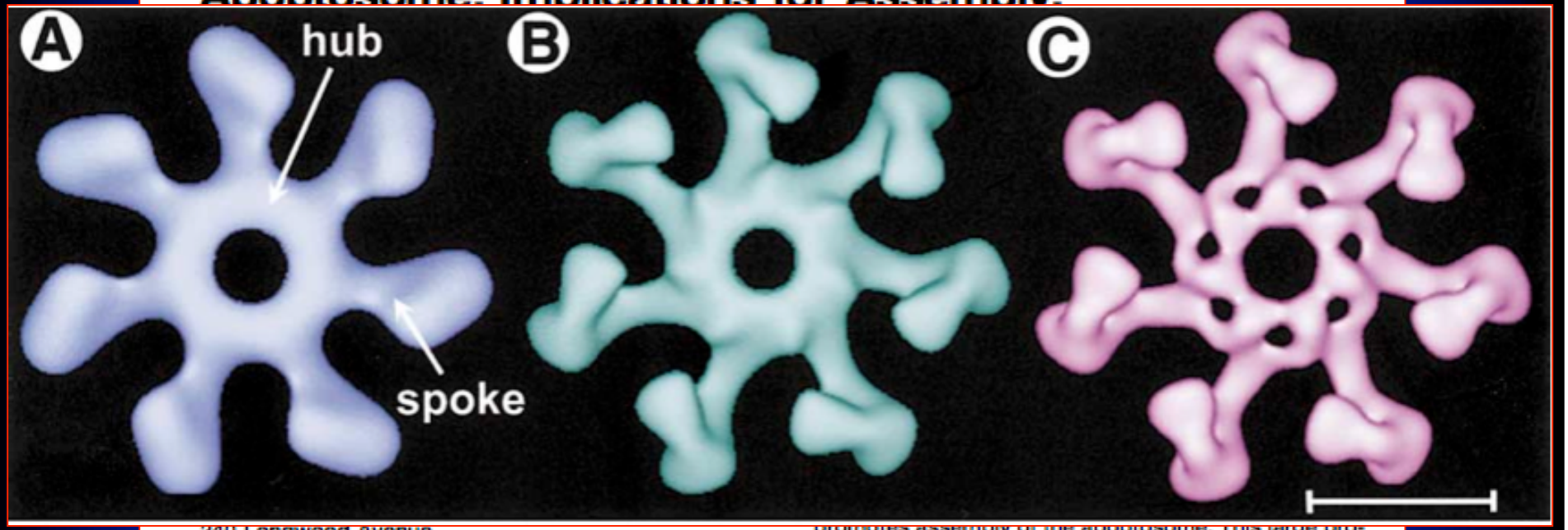
Electron Transport



Protecting Mitochondria

Molecular Cell, Vol. 9, 423–432, February, 2002, Copyright ©2002 by Cell Press

Three-Dimensional Structure of the Apoptosome: Implications for Assembly



240 Longwood Avenue
Boston, Massachusetts 02115
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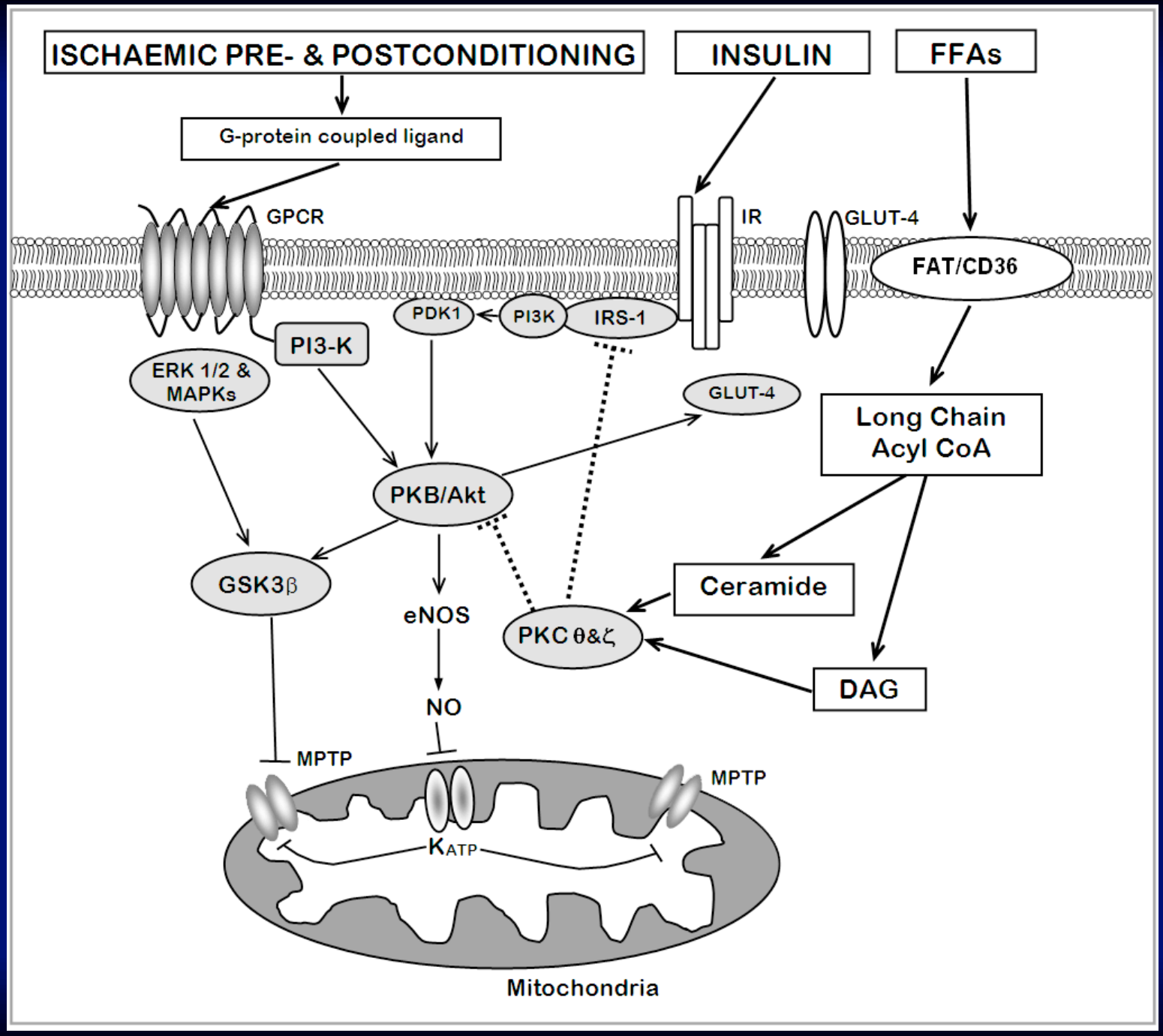
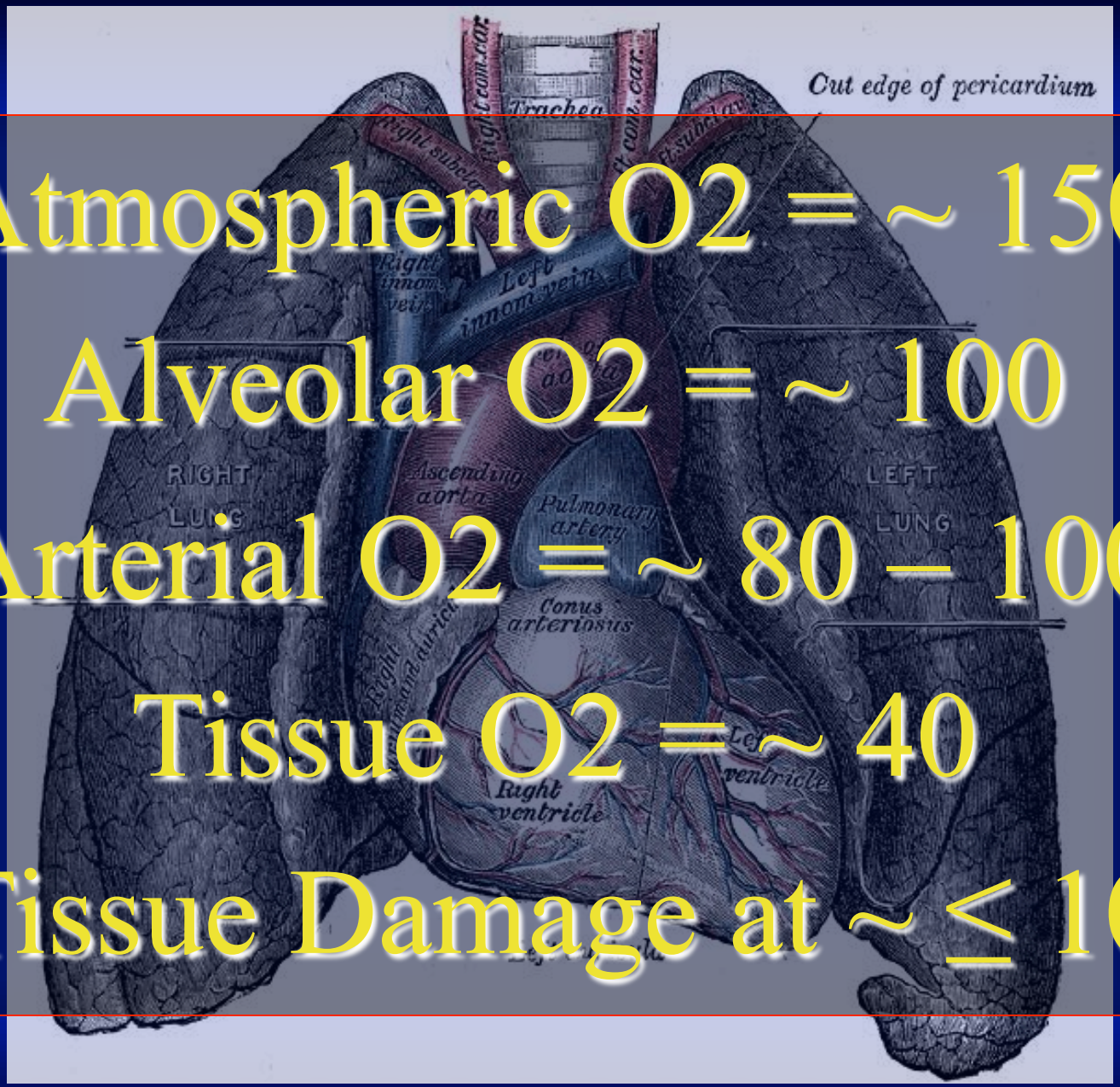
Washington University School of Medicine
660 South Euclid Avenue
St. Louis, Missouri 63110

promotes assembly of the apoptosome. This large protein complex then binds and activates procaspase-9 (Srinivasula et al., 1998; Zou et al., 1997, 1999; Li et al., 1997; Hu et al., 1999). A nonhydrolyzable ATP analog (ADP-CP) also promotes apoptosome formation. This suggests that assembly may be initiated by nucleotide binding rather than hydrolysis (Jiang and Wang, 2000).


ISCHAEMIC PRE- & POSTCONDITIONING
INSULIN
FFAs
G-protein coupled ligand
GPCR
IR
GLUT-4
FAT/CD36
PDK1
PI3K
IRS-1
PI3-K
ERK 1/2 & MAPKs
GLUT-4
Long Chain Acyl CoA
PKB/Akt
GSK3β
eNOS
Ceramide
PKC θ&ζ
DAG
NO
MPTP
MPTP
KATP
Mitochondria

Oxygen Management

Considerations on the movement of oxygen from the atmosphere into the tissues of the body



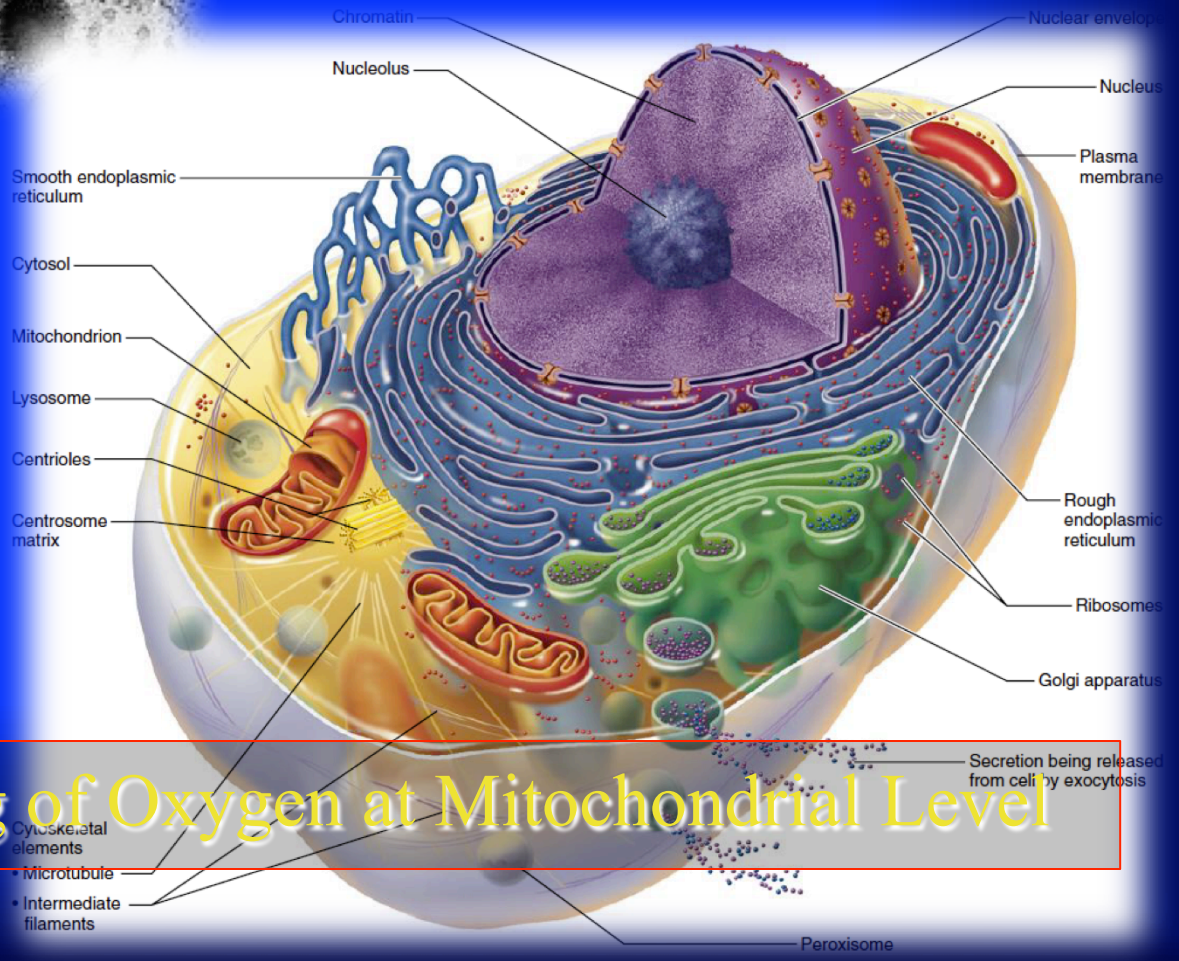
Atmospheric O2 = ~ 150

Alveolar O2 = ~ 100

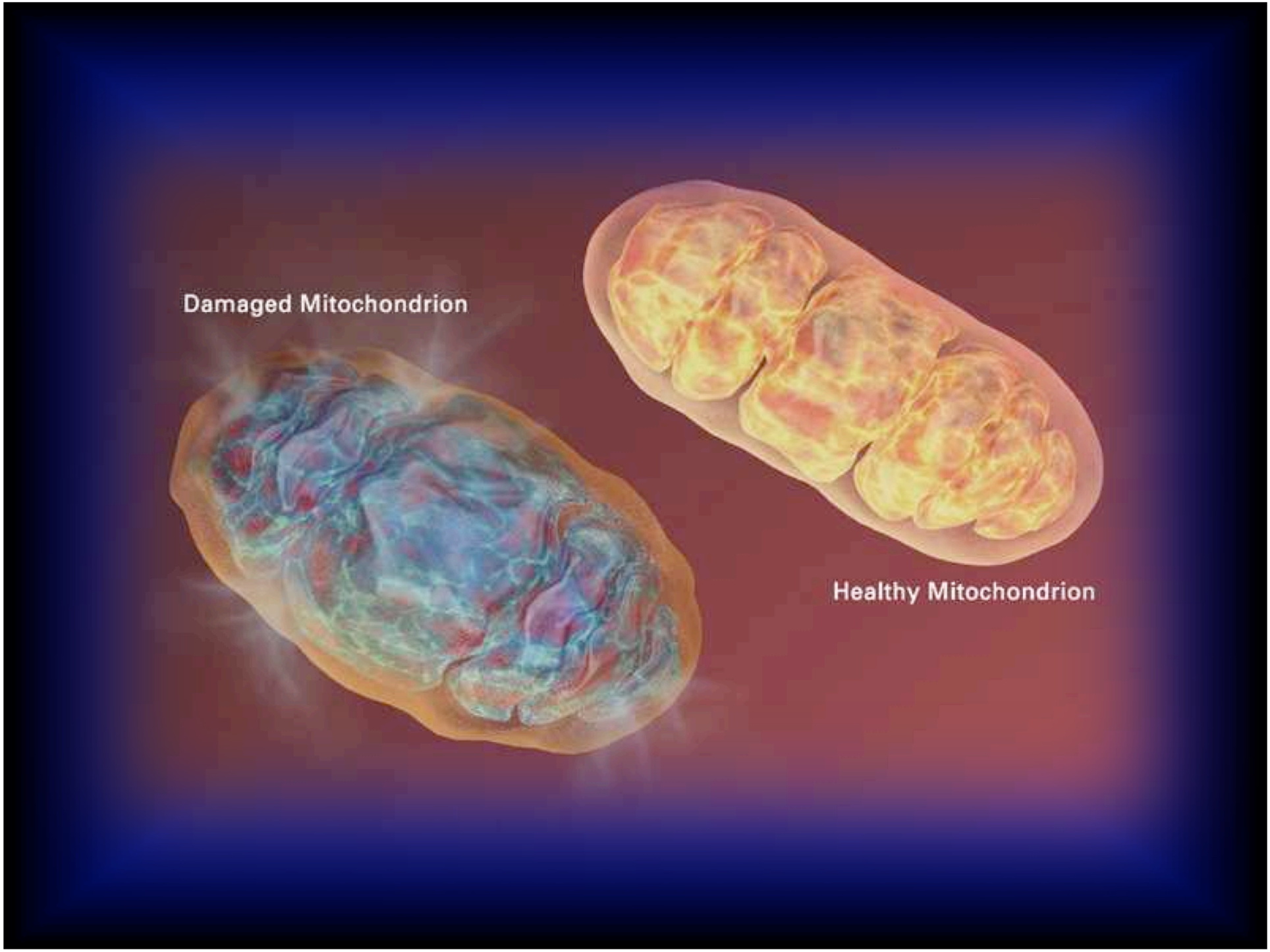
Arterial O2 = ~ 80 – 100

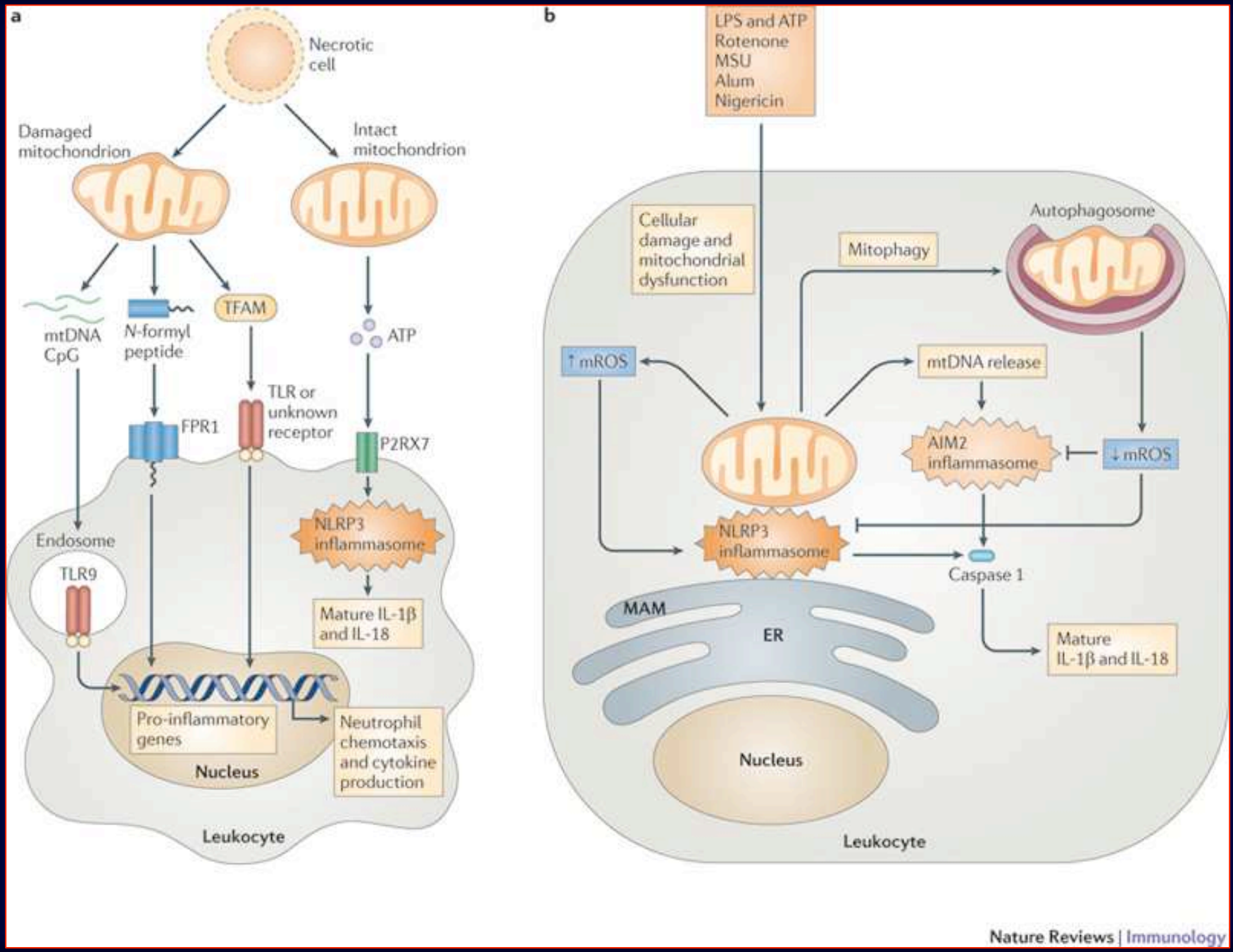
Tissue O2 = ~ 40

Tissue Damage at ~ ≤ 10



40 mm Hg of Oxygen at Mitochondrial Level


Damaged Mitochondrion
Healthy Mitochondrion

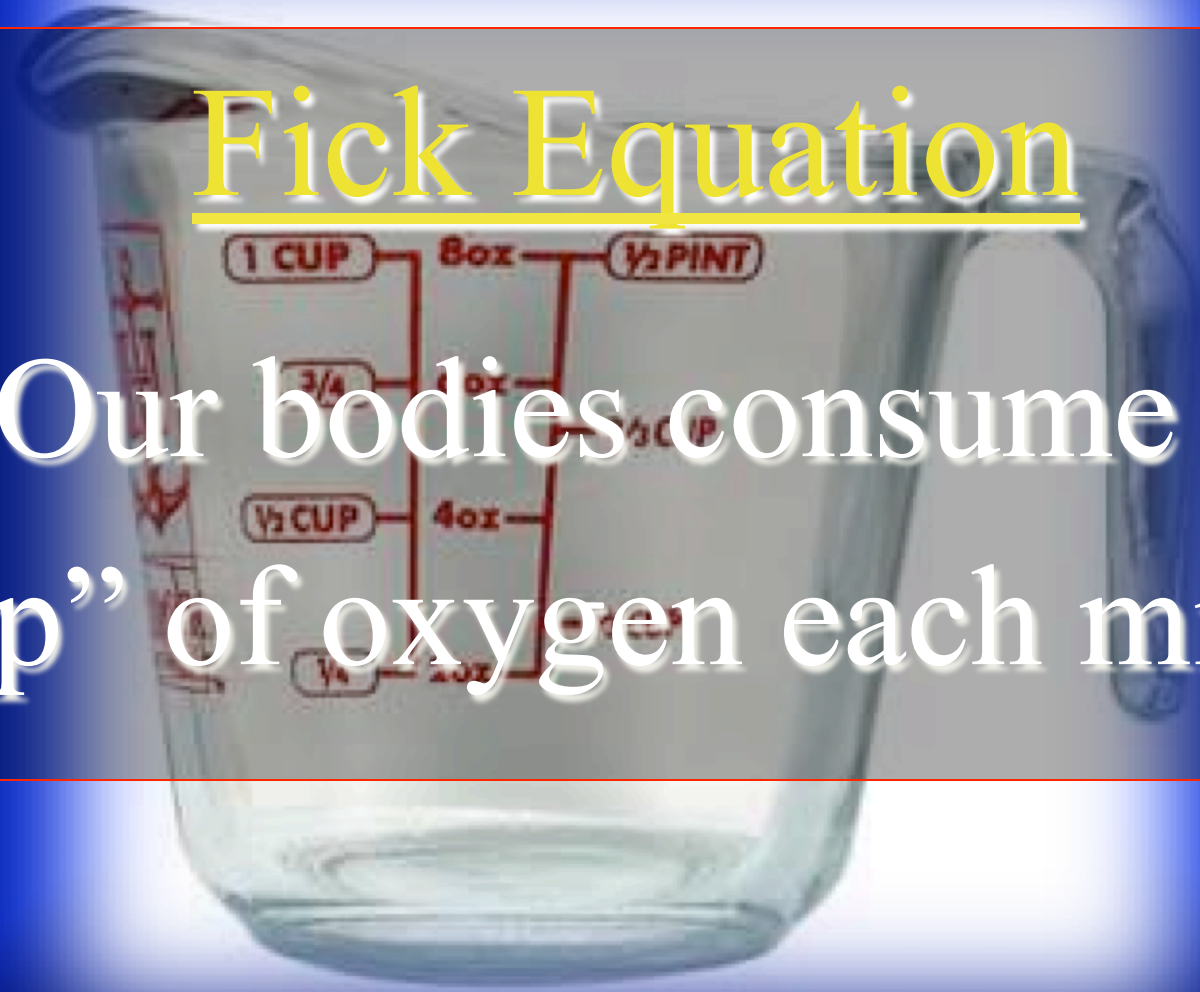

a
Necrotic cell
Damaged mitochondrion
Intact mitochondrion
mtDNA CpG
N-formyl peptide
TFAM
ATP
FPR1
TLR or unknown receptor
P2RX7
Endosome
TLR9
NLRP3 inflammasome
Mature IL-1β and IL-18
Pro-inflammatory genes
Nucleus
Neutrophil chemotaxis and cytokine production
Leukocyte
b
LPS and ATP
Rotenone
MSU
Alum
Nigericin
Cellular damage and mitochondrial dysfunction
Mitophagy
Autophagosome
↑ mROS
mtDNA release
AIM2 inflammasome
↓ mROS
NLRP3 inflammasome
Caspase 1
MAM
ER
Mature IL-1β and IL-18
Nucleus
Leukocyte
Nature Reviews | Immunology

Fick Equation

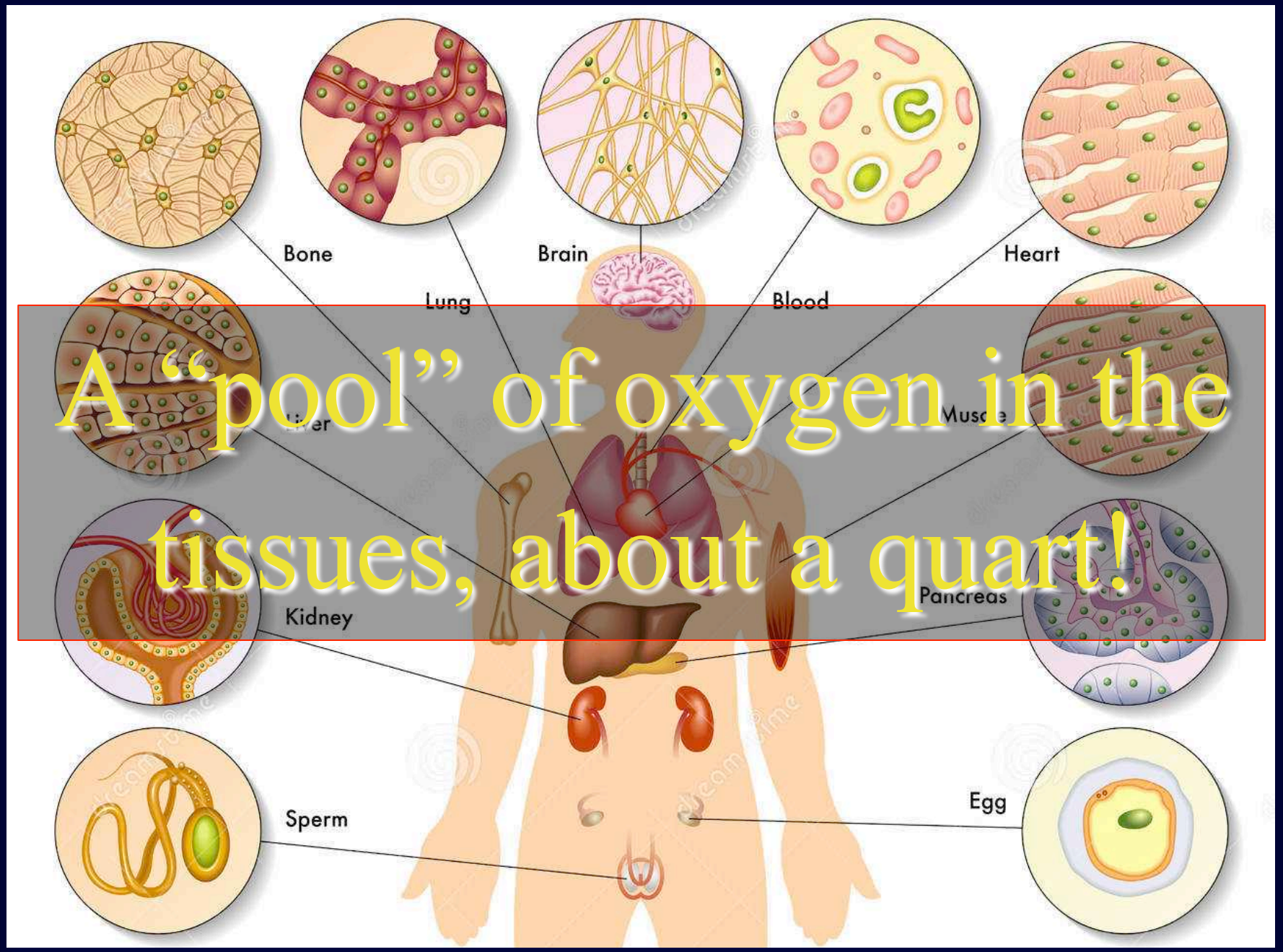
- Gives "consumed oxygen"
- $VO_2 = 1.38\ (Hb)(CO)\ (SaO_2 - SvO_2)/10$

(normally 240-290 cc/min)

Fick Equation



Our bodies consume a "cup" of oxygen each minute

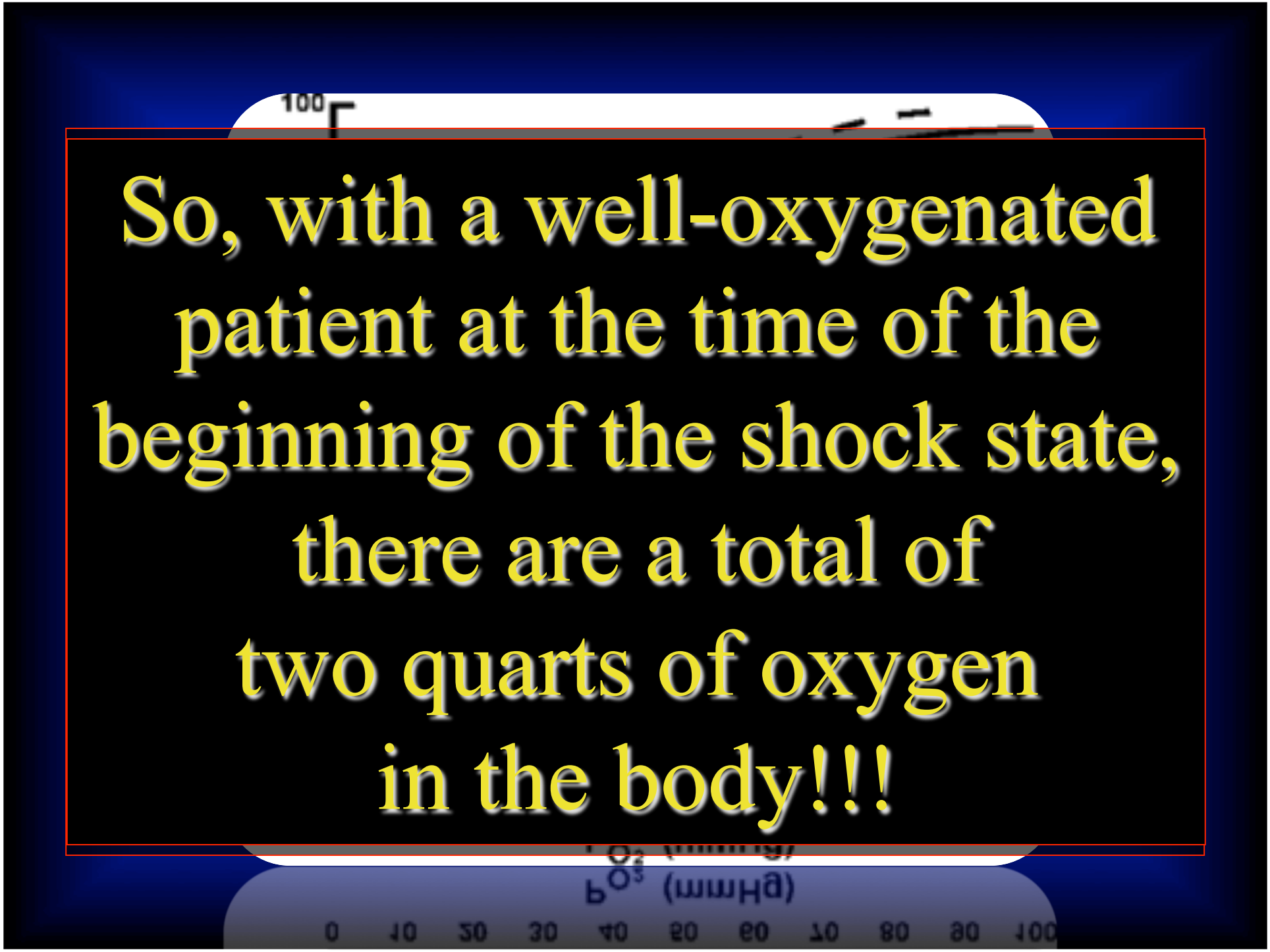


A "pool" of oxygen in the tissues, about a quart!

20 cc O_2 /100 cc Blood

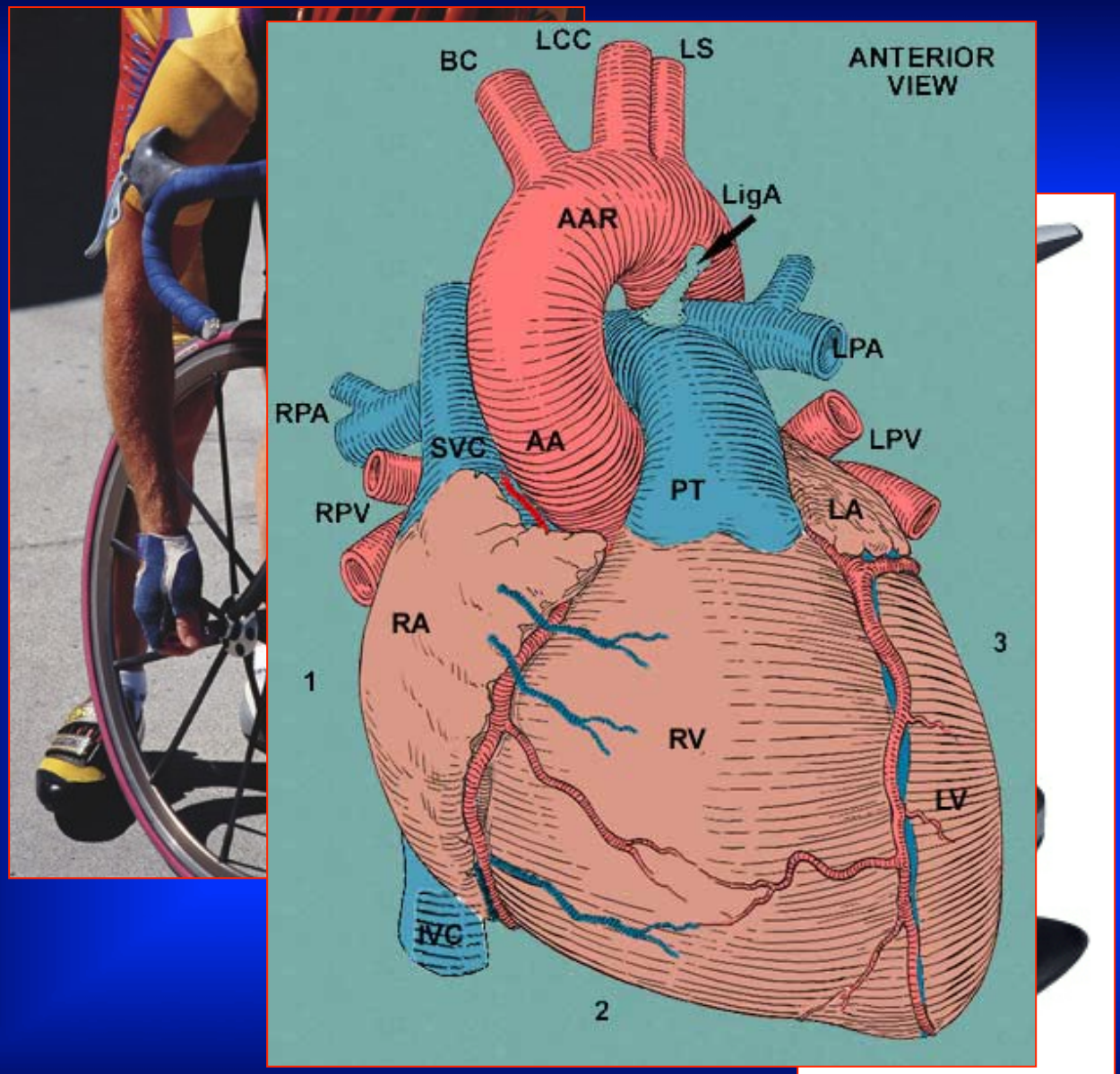
5000 cc / 100 cc = 50 factor

20 cc x 50 = ***A Quart!***


So, with a well-oxygenated patient at the time of the beginning of the shock state, there are a total of two quarts of oxygen in the body!!!


BP = C.O. x PVR
C.O. = HR x SV


BP = C.O. x PVR
C.O. = HR x SV


BC
LCC
LS
ANTERIOR VIEW
LigA
AAR
LPA
RPA
SVC
AA
LPV
PT
RPV
LA
RA
3
1
RV
LV
IVC
2

What does a low blood pressure mean?

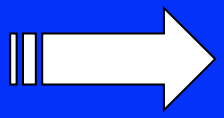
Either...

Or a combination of any of these

…from BTLS/ITLS, editions 2, 3, 4, 5, 6, 7, and 8 Fowler et al

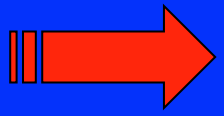
Signs of Shock

Early



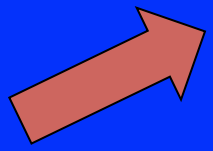
Weak, thirsty, lightheaded
Pale, then sweaty
Tachycardia
Tachypnea
Diminished urinary output

Late



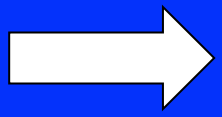
Hypotension
Altered LOC
Cardiac arrest
Death

Shock



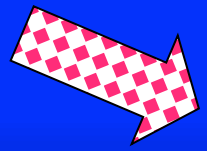
Cardiogenic

Rapid pulse
Distended neck veins
Cyanosis



Volume Loss

Rapid pulse
Flat neck veins
Pale



Vasodilatory

Variable pulse
Flat neck veins
Pale or pink

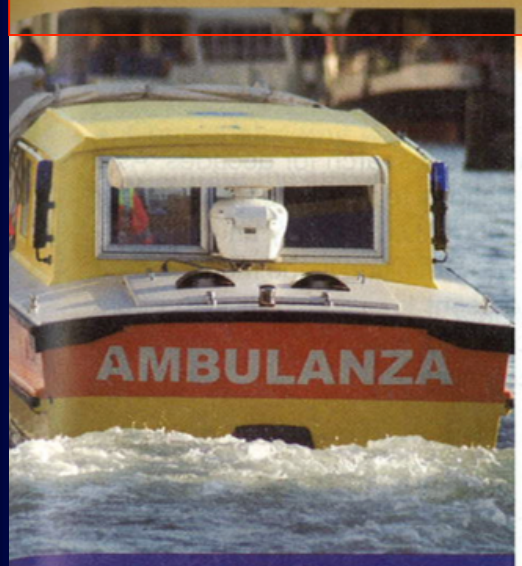
Signs of Shock

Early → *Lactate Begins to appear during this period!!*

Late → **Hypotension**
Altered LOC
Cardiac arrest
Death

ITLS Shock 2015 and Beyond

8



Shock

Raymond L. Fowler, MD, FACEP
Paul E. Pepe, MD, MPH, FACEP, FCCM
John T. Stevens, EMT-P
Mario Luis Ramirez, MD, MPP
Howard Mell, MD, MPH

Shock	Shock	Choc	Wstrząs	Schock
Šok	الصدمه	šok	Šok	Sokk

1. Shock Assessment: How low can you go?

ROC Hypo Resus Pilot Trial of 191 patients with major trauma: No clear indicators!

2. TXA now recommended for traumatic hemorrhage. It is being studied for TBI.

What do we still not know about Tranexamic Acid?

Whether TXA has any impact on trauma outcomes when damage-control resuscitation or MT protocols are used;

The mechanism by which TXA reduced mortality in trauma in the CRASH-2 Trial;

Whether fibrinolysis testing should be performed before consideration of TXA treatment;

What is the optimal dose and timing of TXA in trauma;

Whether other antifibrinolytic agents could be substituted for TXA use in trauma;

Whether TXA is associated with higher seizure rates in trauma or TBI patients.

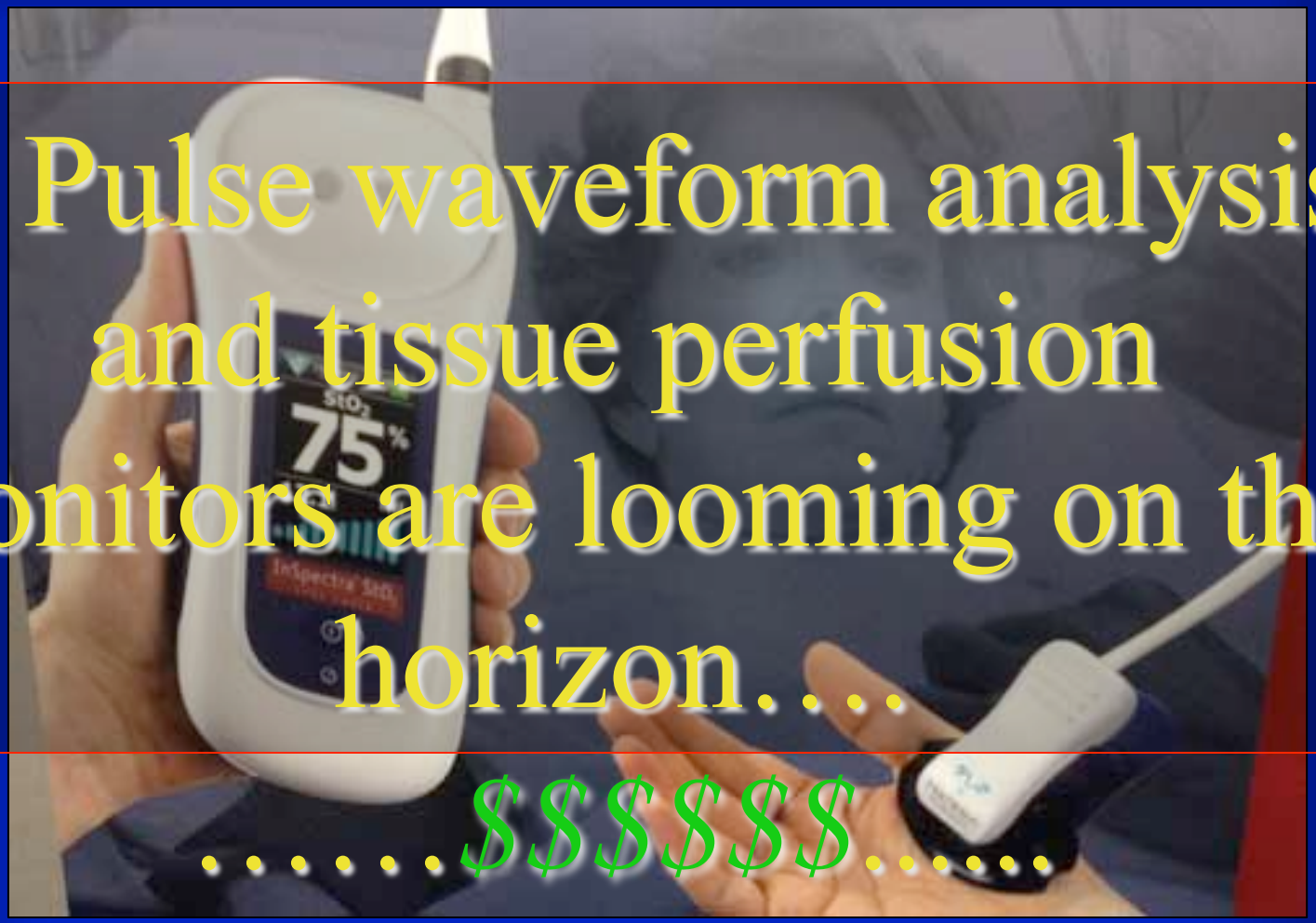
A Rational Approach for TXA use in Trauma

In adult trauma patients with severe hemorrhagic shock (SBP $\leq$ 75 mm Hg), with known predictors of fibrinolysis, or with known fibrinolysis by Thromboelastography (TEG);

ONLY administer TXA if less than 3 hours from time of injury; LATER IS DANGEROUS!!

TXA administration: 1 g intravenously administered over 10 minutes, then 1 g intravenously administered over 8 hours.

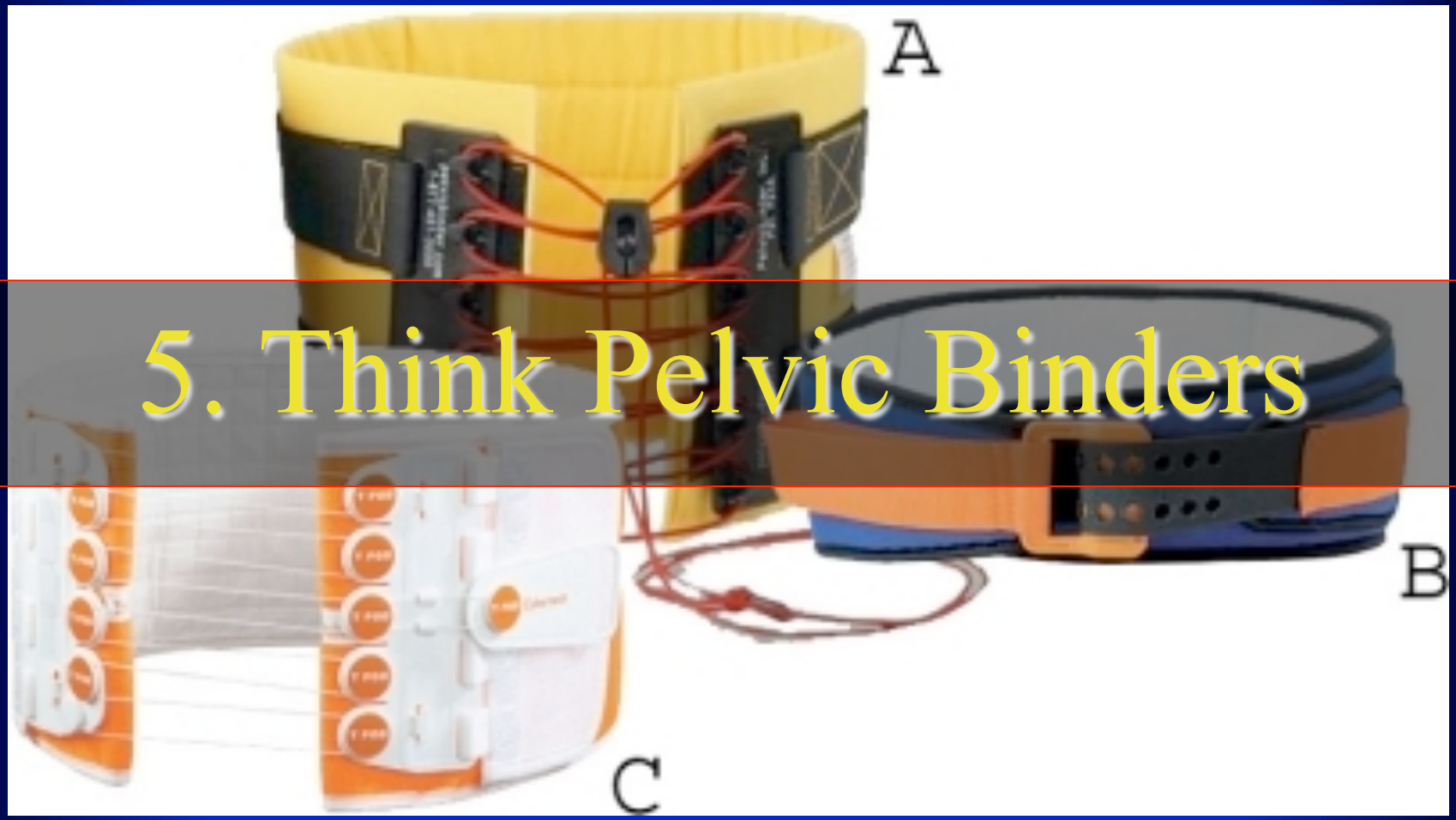
3. Corollary:
The injured brain does NOT tolerate hypotension!



4. Pulse waveform analysis and tissue perfusion monitors are looming on the horizon….

…….$$$$$$……

5. Think Pelvic Binders



6. Intermediary Metabolism stimulation

Surgery. 1987 Sep;102(3):515-27.

Increasing survival of dogs subjected to hemorrhagic shock by administration of fructose 1-6 diphosphate.

Markov AK, Terry J 3rd, White TZ, Didlake RH, Hellems HK.

Abstract

Previous reports from this laboratory described animal experiments in which intravenous administration of fructose 1-6 diphosphate (FDP) at the onset of hypovolemia, toxemia, and trauma effected improvement in hemodynamic and metabolic parameters, attenuation of tissue damage, and a significant increase in survival. The obvious question remained: Would this agent be as effective if administered after the onset of the shock syndrome? Thus 72 anesthetized dogs were subjected to normotensive hemorrhagic shock and were subsequently treated with FDP at 30 minutes, 1 hour, 90 minutes, and 2 hours after exsanguination. Analysis of the results (as compared with vehicle-treated controls) revealed evidence of improved cardiac output and arterial pressure (p less than 0.02), conservation of effective circulatory volume, better oxygen utilization, and a significant increase in survival (p less than 0.0001). These results, in conjunction with earlier experimental and recent clinical data, indicate that the therapeutic effect of FDP in ischemic and hypoperfusion states is in part metabolically mediated by the augmentation of carbohydrate utilization. Prevention of tissue injury is in part due to the inhibition of generation of oxygen-derived free radicals by neutrophils.

Is there a future in trauma resuscitation for intermediary mediator modulators??

- *FDP*
- *Vitamin C*
- *Nitric Oxide inhibitors*


7. Serum Lactate Levels may be helpful….though remember Dr. Markov!!

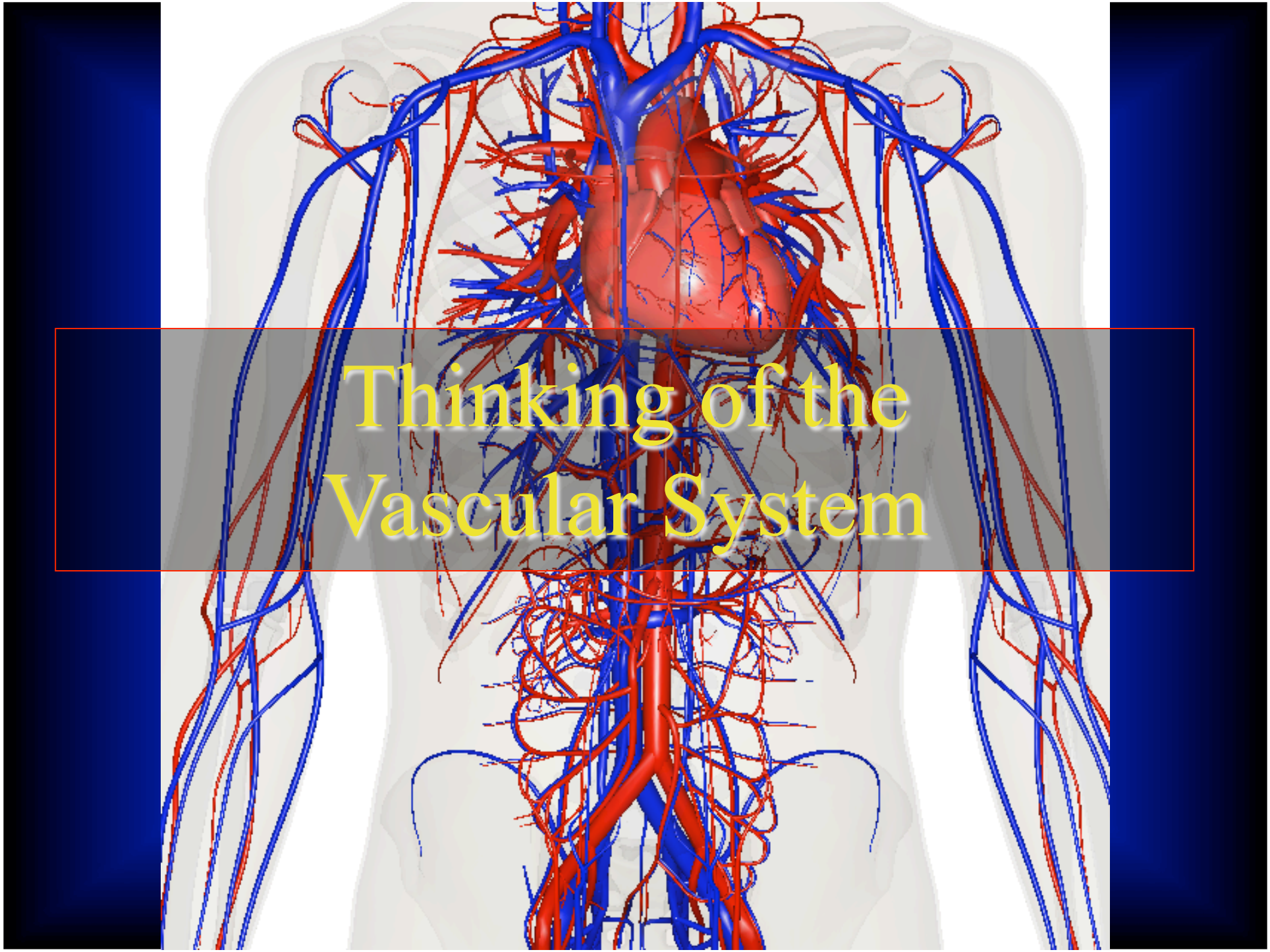
The Current Evidence:

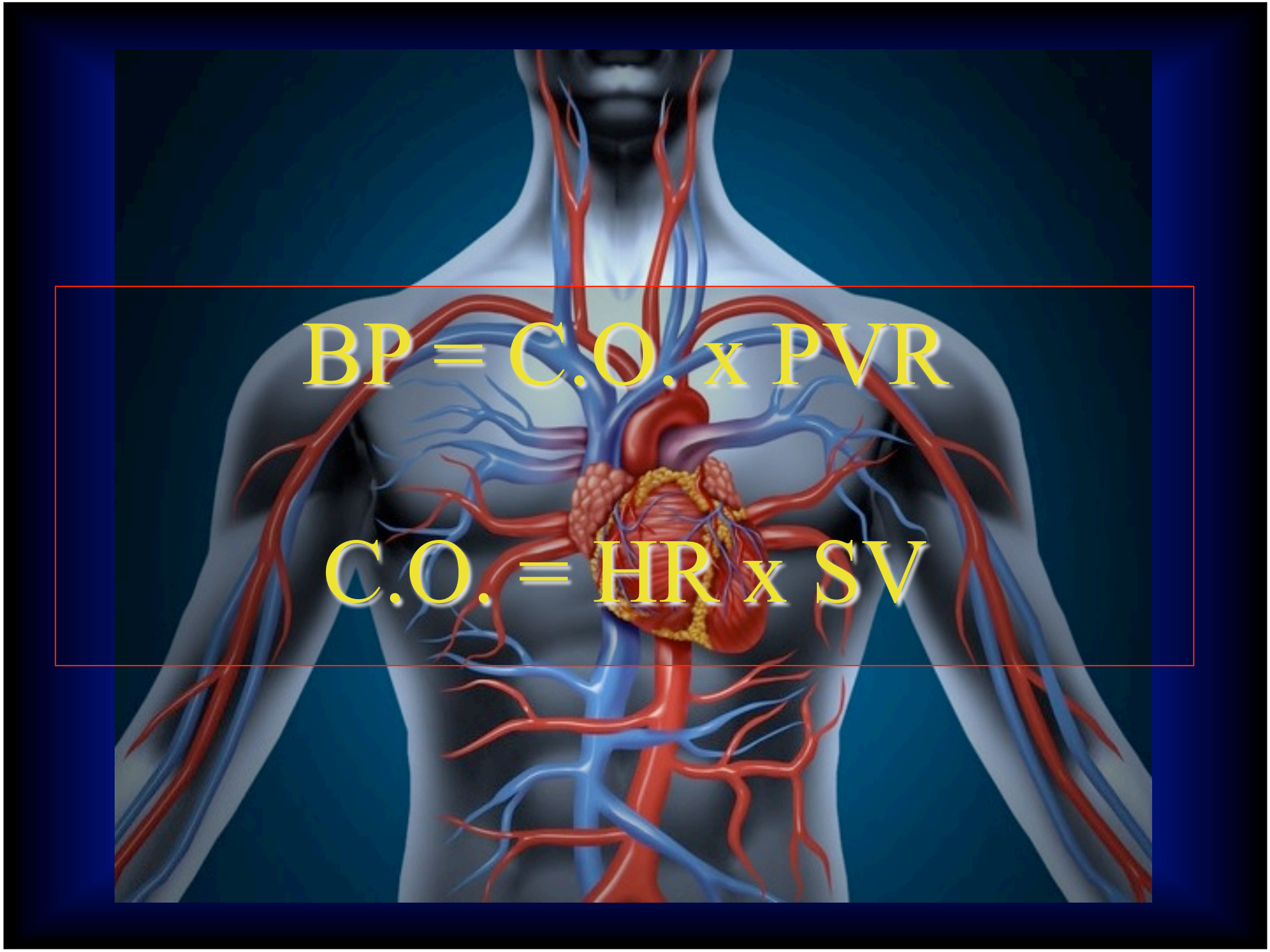
Reaching the plane of volume resuscitation vs. worsening outcomes

The Current Evidence:

Time-Sensitive Condition System Construction: Can every facility provide optimal care?

Frequency? Training?


Thinking of the
Vascular System


BP = C.O. x PVR
C.O. = HR x SV

It is with attention to all of the elements of the vascular system that evaluation and treatment are optimized

The Approach to the Hypotensive Patient in the Modern Era

Used with gratitude from www.emdocs.net

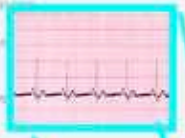
A Systematic Approach to arriving at the Best Possible Explanation

Pace or Cardiovert?

unstable brady (<40)

unstable, non-sinus tachy (>170)

1. HEART RATE



2. VOLUME

U/S for IVC & LV volume

hypovolemia?

hemorrhage? → FAST, AAA?

**IVC >>> LV volume if there is obsxn to flow through heart or lungs ...

Tamponade?

New RV infarction?

Massive PE?

Asthmatic alveolar air trapping?

Tension pneumothorax?

** When 1-3 are okay, then volume is going forward ... so consider peripheral vasodilation +/- mitochondrial dysfxn

Cervical spinal cord compression?

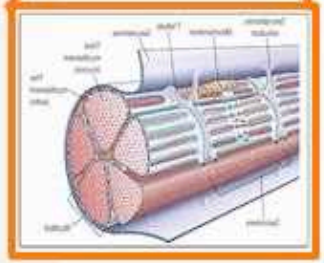
Anaphylaxis?

Fulminant liver failure?

Sepsis?

Mitochondrial poison (Cyanide, CO)?

3. CONTRACTION



ECG & U/S for LV fxn

Volume in lumen during diastole?

LV walls collapse well in systole?

Is volume truly going forward?

(listen for regurg murmur & rales)

1. **Heart Rate (HR):**

- Approach: Look at the HR on the monitor.
- **Increased or decreased HR can lead to hypotension.**
- **Tachycardia:** In general, unstable, non-sinus HR > 170 (threshold varies with compliance of LV and age of pt) can result in hypotension.
  - Physiology:
    - ↑ HR -> ↓ stroke volume because there is **less time for diastolic filling** -> ↓ Cardiac Output (HR x SV) -> hypotension
    - Additionally, since less time is spent in diastole, the ventricular myocardium has **inadequate relaxation time** to allow flow through *intramyocardial* perforating coronary branches. This leads to **ischemia of the LV subendocardium**. Ischemia can progress from the subendocardium to more superficial layers of the LV myocardium and result in **poor contractile function** -> hypotension
    - Also, diffuse ischemia can produce heterogeneous myocyte repolarization patterns -> **re-entrant arrhythmia or fibrillation**.
  - The key is to differentiate between tachycardia as a **response to hypotension** or tachycardia as the **cause of hypotension**.
    - Typically HR > 170 starts to affect diastolic filling and can result in hypotension -> suspect tachycardia as the cause of hypotension.
    - Typically HR < 170 does not affect diastolic filling -> suspect tachycardia as the response to hypotension.
      - Of note: AFib w/ RVR may result in hypotension at lower HR thresholds because short diastolic filling is compounded by loss of atrial contraction, and hence LV filling is solely passive. In this setting, non-compliant LVs may fail to fill at HRs as low as 140.
- **Bradycardia:** In general, unstable HR < 50 can result in hypotension.
  - Physiology: ↓ Heart Rate -> ↓ Cardiac Output -> Hypotension
- Differential: Know your tachyarrhythmias and bradyarrhythmias, but in general treat appropriately -> cardiovert or pace the patient.

2. **Volume Status:**

- Assess this **after** you've determined that the HR is a result of hypotension, not the cause (since rapid heart rates impair diastolic filling and will enlarge the IVC as volume remains peripherally).
- Physiology: ↓ Volume Status -> ↓ Stroke Volume -> ↓ Cardiac Output -> Hypotension
- Approach: Do a quick bedside physical examination, then grab your ultrasound probe to assess the **inferior vena cava (IVC) and left ventricle (LV) volume.**
  - Physical examination:
    - Look at the patient's face.
      - Dry mucous membranes?
      - Sunken eyes?
    - Feel the extremities.
      - Abnormal skin turgor (ie tenting when pinched)?
      - Poor capillary refill (ie > 2 seconds)?
      - Weak pulse?
      - Cool extremities?
  - IVC/LV Ultrasound

1. **Flat/collapsed IVC** (anteroposterior diameter of the IVC < 2 cm in size with > 50% collapse with respiratory variation) with **hyperdynamic LV.**
   - Differential: **Hypovolemia vs. Hemorrhage.**
     - Is this due to hypovolemia from dehydration?
     - Hemorrhage from a ruptured AAA/ectopic/GI Bleed?
       - FAST the patient
         - Abdominal aorta > 3 cm indicates AAA
         - Abdominal or pelvic free fluid
       - Serial bedside HGB testing
     - Treatment: Fluid/blood resuscitation
2. **Plethoric / plump, non-compressible IVC,** which is **significantly greater than LV volume.**
   - Ask yourself if there is an obstruction to the flow through the heart or the lungs? -> Obstructive shock
     - Differential: Cardiac tamponade vs. RV infarction vs. severe pulm HTN/massive pulmonary embolism (PE) vs. asthmatic alveolar air trapping vs. tension pneumothorax (PTX)
       - Ultrasound findings to look for:
         - Anechoic fluid surrounding heart with diastolic collapse -> cardiac tamponade
         - RV strain (RV/LV ratio > 0.9, "D sign") -> Pulmonary HTN of some cause (massive PE? Alveolar air trapping?)
         - Lack of lung sliding or "bar code" sign -> PTX
       - Treatment: Depends on underlying cause
3. **Distended, non-collapsing IVC with dilated LV**
   - Move on to cardiac cause in step 3
4. **Normal IVC with diameter >2cm but <50% collapse with respiration**
   - May trial a small bolus of volume if lung auscultation is clear
   - Move on to cardiac cause in step 3

3. **Cardiac Performance:**

- Assess this **after** you've determined that step 1 and 2 are normal. That is, the HR is not the cause of diastolic limitations to myocardial blood flow, intravenous volume is adequate, and there is no obstruction to flow through the thoracic circuit.
- Physiology: ↓ Cardiac Contractility -> ↓ Stroke Volume -> ↓ Cardiac Output -> Hypotension
- Approach: Assess **myocardial performance** by cardiopulmonary physical examination, ECG, and bedside ultrasound to assess left ventricular (LV) function.
  - Physical examination
    - High-pitched holosystolic murmur radiating to axilla?
    - Diastolic murmur at RUSB radiating to LLSB?
    - Systolic murmur at RUSB radiating to neck?
    - Crackles/rales?
    - Elevated JVP or obvious JVD?
    - Peripheral edema?
  - Ultrasound – Short axis parasternal view
    - Do the LV walls collapse well in systole?
      - If not, there is decreased LV contractility/EF
        - Treatment: inotropy to move blood forward +/- vasopressors to increase SVR/diastolic pressure and hence coronary perfusion.
      - Is volume truly going forward? -> Acute mitral regurgitation (MR) or acute aortic regurgitation (AR)
        - Murmur and rales on exam with multiple B lines on lung sonography
- Differential: **Cardiogenic shock** (Decompensated heart failure, acute MI, acute MR, acute AR)

4. **Systemic Vascular Resistance:**

- If 1-3 are okay, then the pt is hypotensive *despite* a HR allowing diastolic filling of LV lumen and coronary arteries, adequate intravenous volume, no obstruction through the pulmonary circuit and hence adequate LV diastolic volume, and adequate contractile function with forward flow out the aortic root. In this setting, there must be a component of peripheral vasodilation to explain low intravascular tone at the measured artery.
- Physiology: Since blood pressure is a balance of cardiac output and systemic vascular resistance, decreasing SVR can result in hypotension.
- Approach:
  - Physical examination
    - How do the extremities feel? Are they warm and vasodilated? Or are they cold, clamped and vasoconstricted?
- Differential: **Distributive shock** (sepsis, anaphylaxis, vasodilatory medication overdose, neurogenic shock, fulminant liver failure, and other causes of severe acidemia including toxic ingestion, inherent metabolic disturbance, or mitochondrial poisons like cyanide, hydrogen sulfide, & carbon monoxide).
  - Treatment: Depends on underlying cause

Follow a Four Step Systematic Approach

BP = C.O. X PVR

1. Heart Rate
2. Volume Status
3. Cardiac Performance
4. Systemic Vascular Resistance

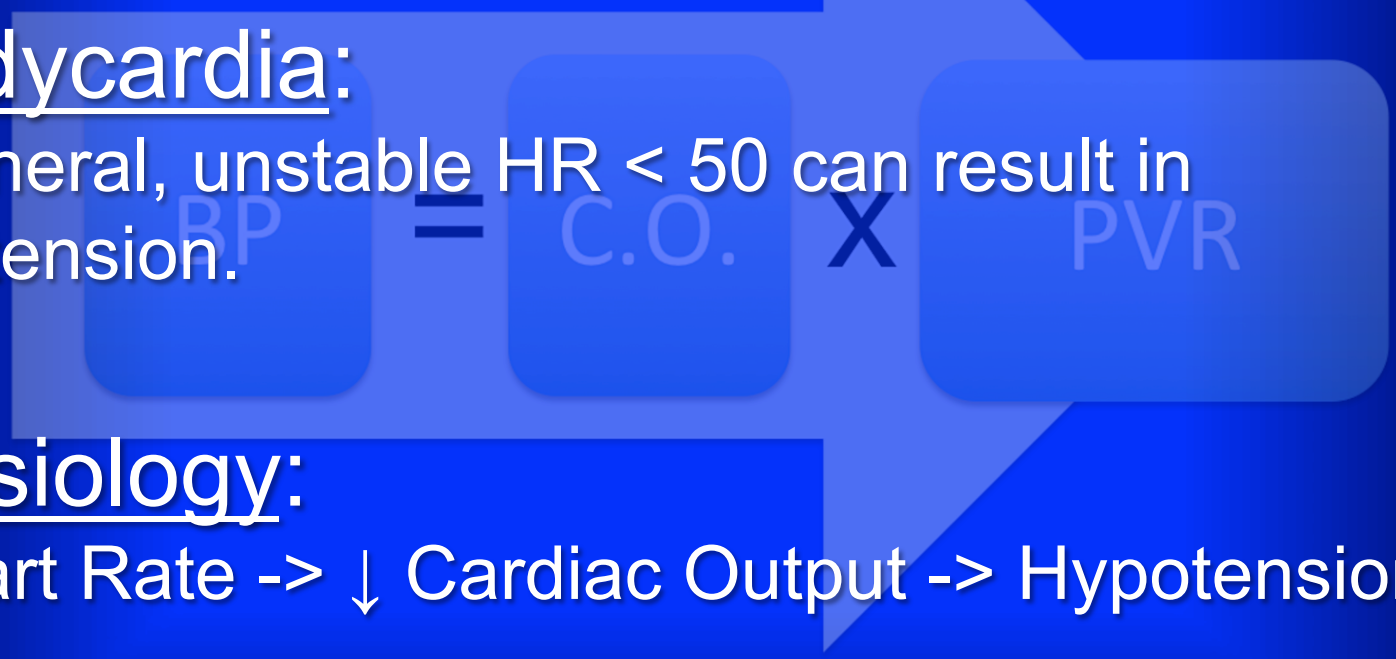
Evaluating Heart Rate as a Cause of Shock

1. ↑ HR -> ↓ stroke volume because there is ***less time for diastolic filling*** -> ↓ Cardiac Output (HR x SV) -> hypotension

Evaluating Heart Rate as a Cause of Shock

2. Since less time in diastole, the ventricular myocardium has inadequate relaxation time to allow flow through *intra-myocardial* perforating coronary branches. *This leads to ischemia of the LV -> poor contractile function -> hypotension*

Evaluating Heart Rate as a Cause of Shock



Bradycardia:

In general, unstable HR < 50 can result in hypotension.

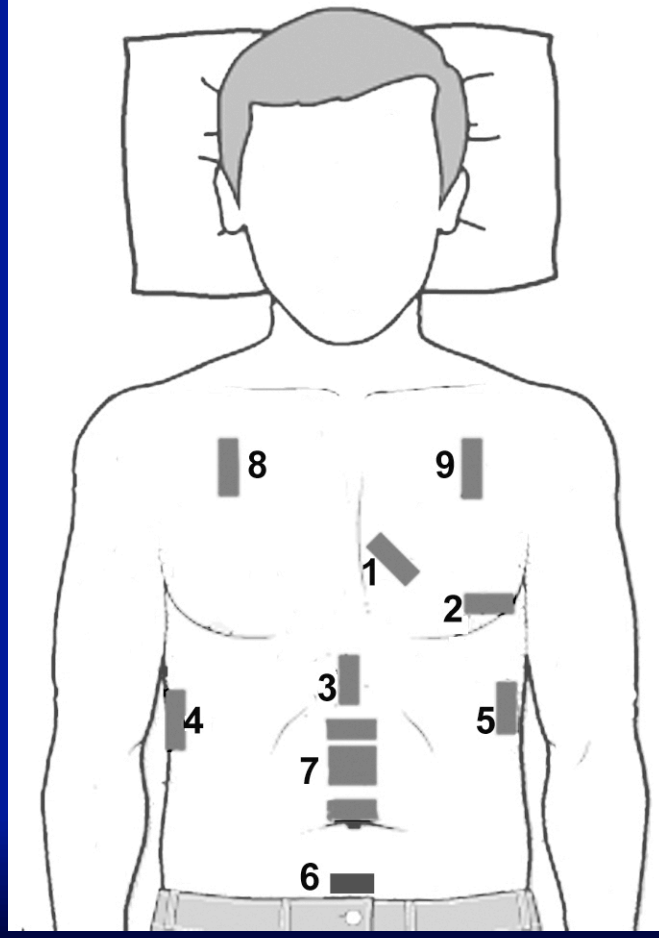
Physiology:

↓ Heart Rate -> ↓ Cardiac Output -> Hypotension

Evaluating Hypovolemia as the Cause of Shock

Physiology: ↓ Volume Status -> ↓ Stroke Volume -> ↓ Cardiac Output -> Hypotension Approach: Do a quick bedside physical examination, then grab your ultrasound probe to assess the **inferior vena cava (IVC) and left ventricle (LV) volume.**

Evaluating Hypovolemia as the Cause of Shock

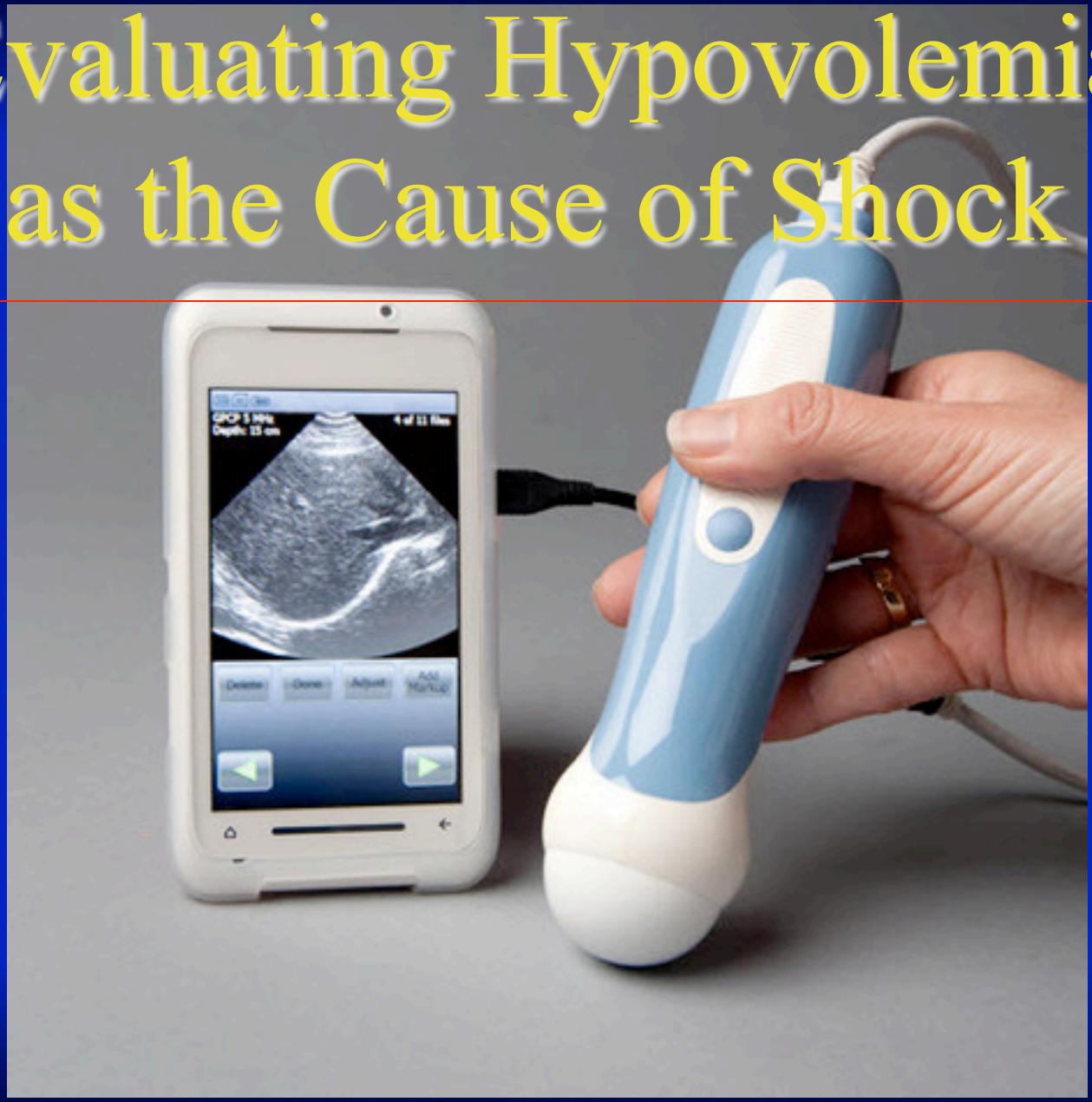


RUSH Exam Sequencing

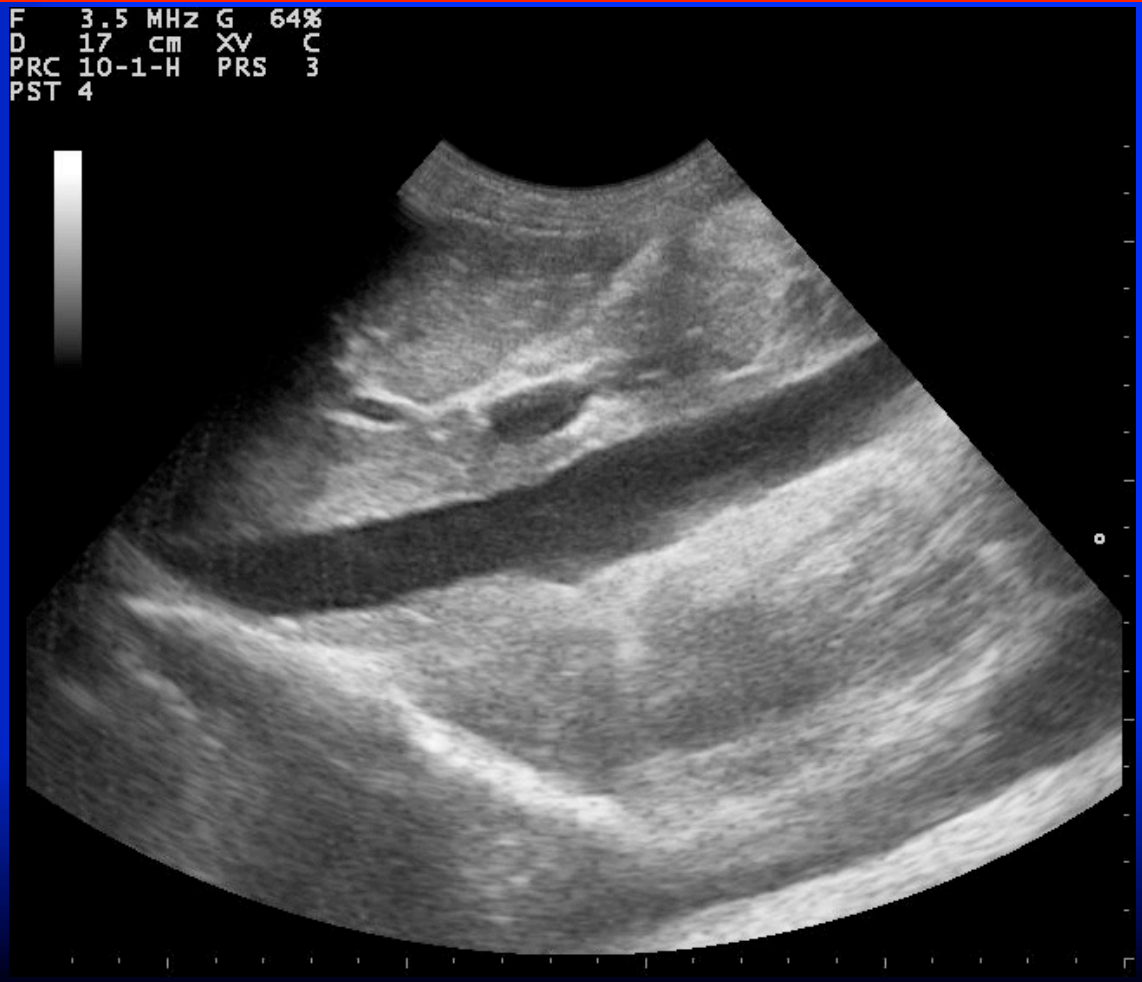
1. Parasternal Long Cardiac View
2. Apical Four-Chamber Cardiac View
3. Inferior Vena Cava View
4. Morison's with Hemothorax View
5. Splenorenal with Hemothorax View
6. Bladder View
7. Aortic Slide Views
8. Pneumothorax View
9. Pneumothorax View

Use Curvilinear Array for 1-7
Use High-Frequency Array for 8 & 9

Evaluating Hypovolemia as the Cause of Shock



Evaluating Hypovolemia as the Cause of Shock



Evaluating Hypovolemia as the Cause of Shock

Physical examination:

- Look at the patient's face.
  - Dry mucous membranes?
  - Sunken eyes?
- Feel the extremities.
  - Abnormal skin turgor (i.e., tenting when pinched)?
  - Poor capillary refill (ie > 2 seconds)?
  - Weak pulse?
  - Cool extremities?

Evaluating Hypovolemia as the Cause of Shock

IVC/LV Ultrasound:

Flat/collapsed IVC (anteroposterior diameter of the IVC < 2 cm in size with > 50% collapse with respiratory variation) with **hyperdynamic left ventricle [LV])**

Differential: **Hypovolemia vs. Hemorrhage.**

Is this due to hypovolemia from dehydration?

Plethoric / plump, non-compressible IVC, which is **significantly greater than LV volume.**

Ask yourself if there is an obstruction to the flow through the thorax ("obstructive, or mechanical, shock")

Evaluating Hypovolemia as the Cause of Shock

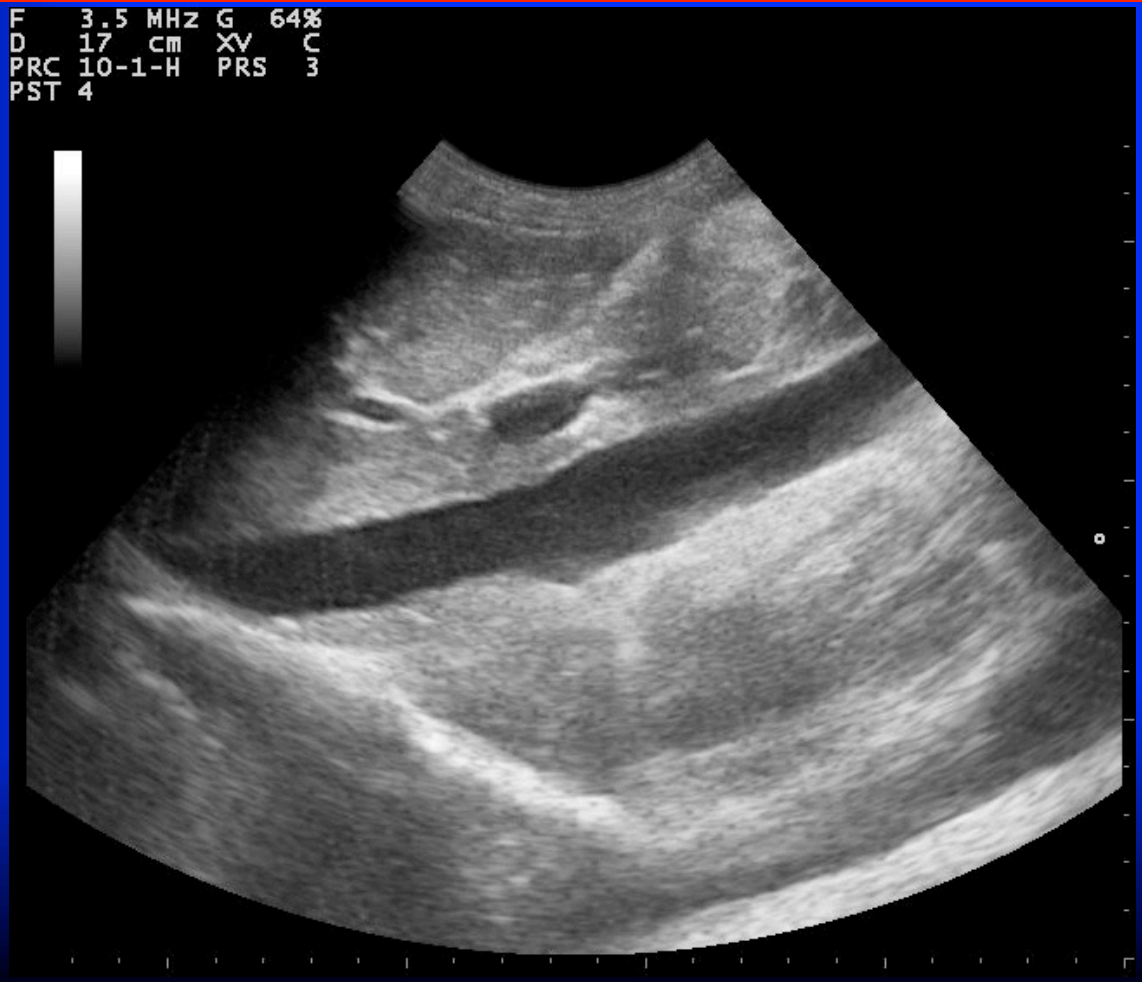
Distended, non-collapsing IVC with dilated LV:

Move on to cardiac cause in step 3

Normal IVC with diameter >2cm but <50% collapse with respiration:

May trial a small bolus of volume if lung auscultation is clear, then Move on to cardiac cause in step 3

Evaluating Hypovolemia as the Cause of Shock



Evaluating Cardiac Performance as the Cause of Shock

Assess this **after** you've determined that step 1 and 2 are normal:

…that is, the HR is not the cause of diastolic limitations to myocardial blood flow, intravenous volume is adequate, and there is no obstruction to flow through the thoracic circuit….

Evaluating Cardiac Performance as the Cause of Shock

Physiology: ↓ Cardiac Contractility -> ↓ Stroke Volume -> ↓ Cardiac Output ->

Hypotension

Evaluating Cardiac Performance as the Cause of Shock

Approach: Assess myocardial performance

- Cardiopulmonary physical examination
- Electrocardiogram
- Bedside ultrasound to assess left ventricular (LV) function

Evaluating Systemic Vascular Resistance as the Cause of Shock

BP = C.O. X PVR

Physiology: Since blood pressure is a balance of cardiac output and systemic vascular resistance, decreasing SVR can result in hypotension.

Evaluating Systemic Vascular Resistance as the Cause of Shock

BP = C.O. X PVR

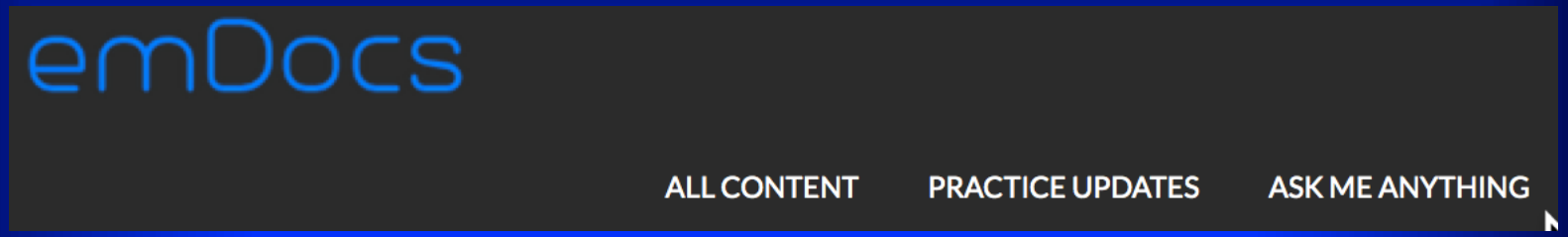
Approach: Physical examination

How do the extremities feel? Are they warm and vasodilated?

Or, are they cold & vasoconstricted?

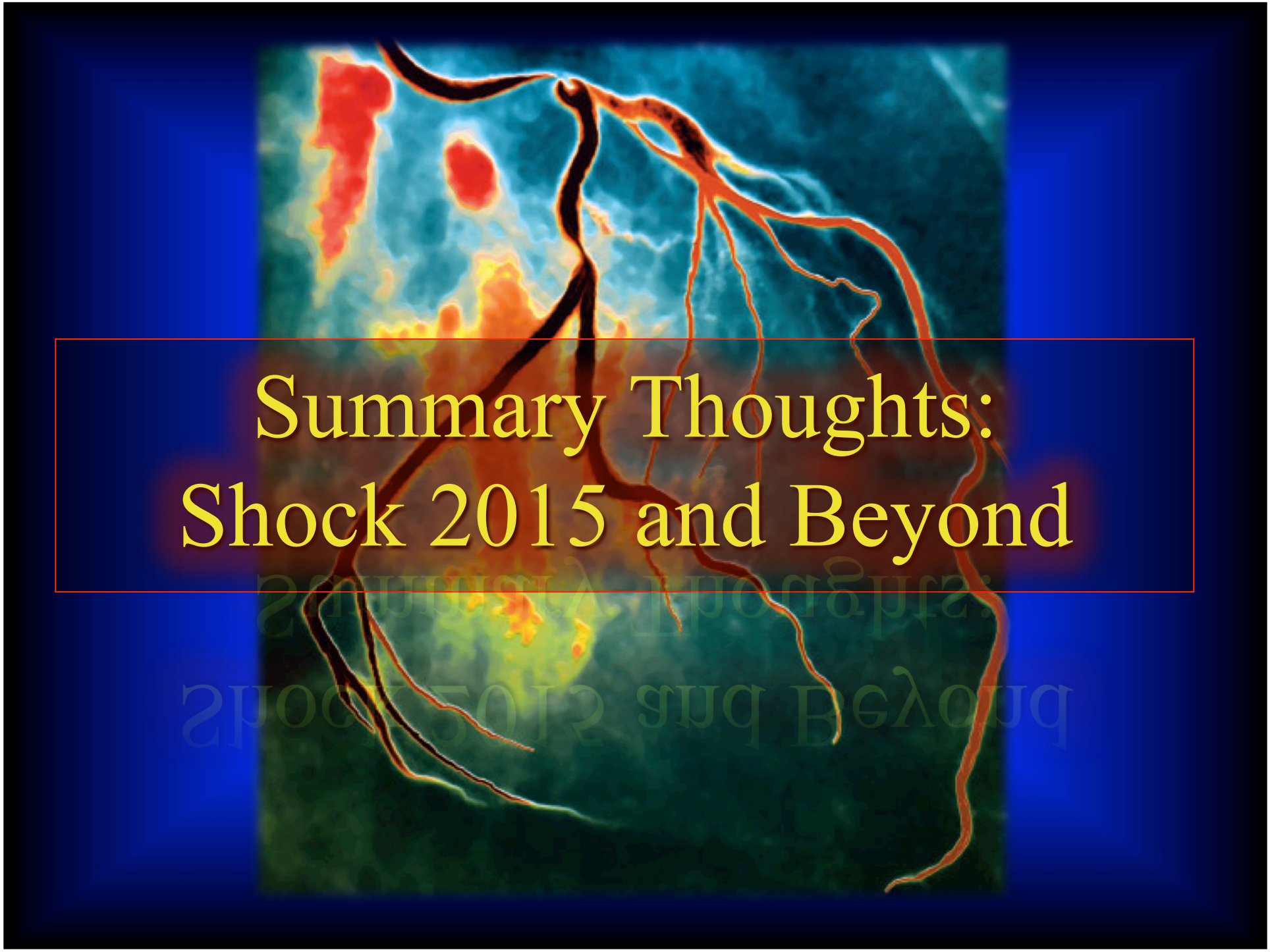
Evaluating Systemic Vascular Resistance as the Cause of Shock

Differential: **Distributive shock** (sepsis, anaphylaxis, vasodilatory medication overdose, neurogenic shock, fulminant liver failure, and other causes of severe acidemia including toxic ingestion, inherent metabolic disturbance, or mitochondrial poisons like cyanide, hydrogen sulfide, & carbon monoxide).



www.emdocs.net

http://www.emdocs.net/hypotensive-ed-patient-sequential-systematic-approach/


Summary Thoughts:
Shock 2015 and Beyond

"There are **known knowns**. These are things we know that we know.

"There are **known unknowns**. That is to say, there are things that we know we don't know.

"But there are also unknown unknowns. There are things we don't know we don't know."

- Donald Rumsfeld

What do we know?

Things we KNOW we KNOW

Things we KNOW that we don't KNOW

Things we don't KNOW that we don't KNOW

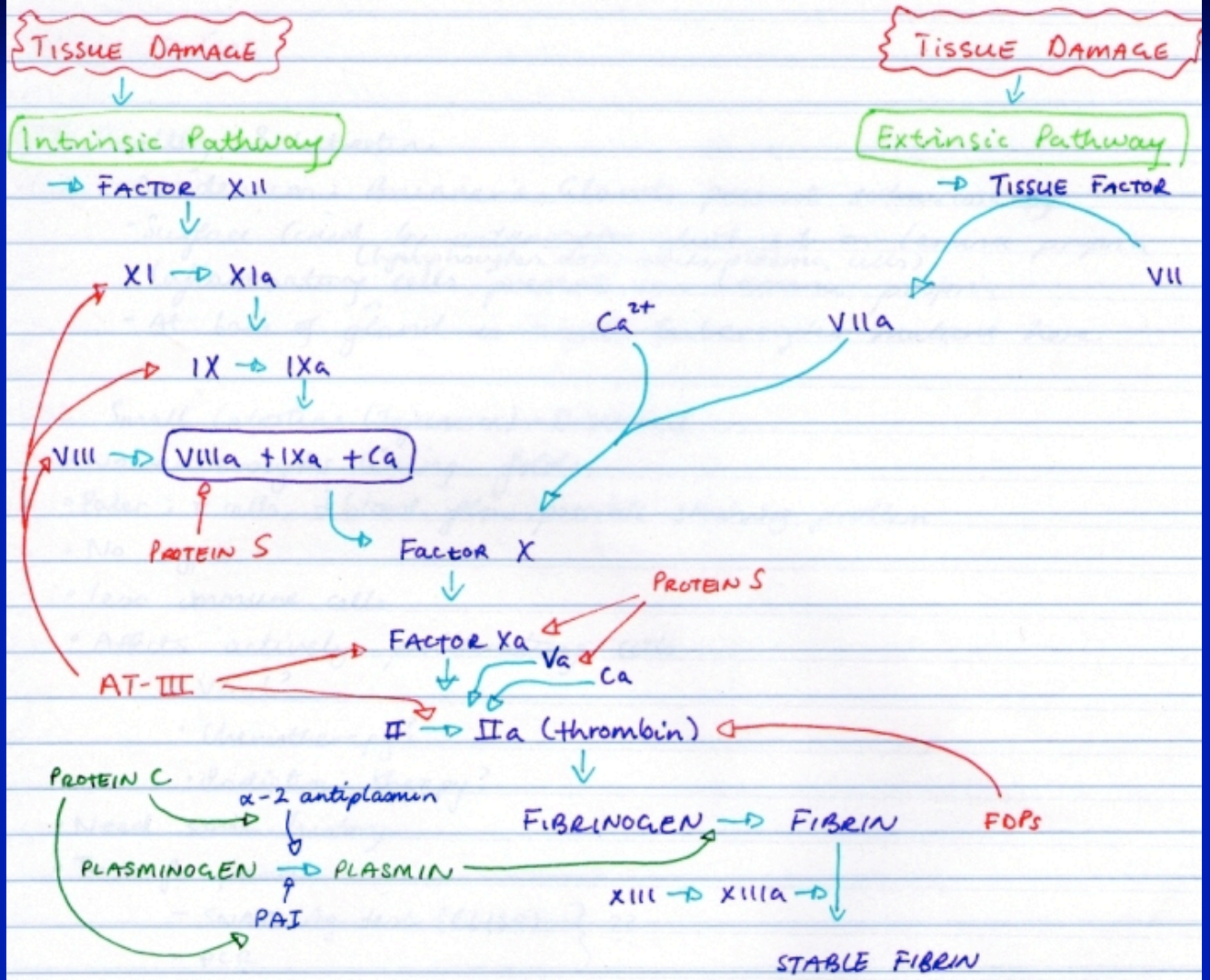
Things we KNOW we KNOW

- *Airway Management*
- *Bleeding Control*
- *Immobilization (probably)*
- *Positive pressure for ARDS*
- *Avoiding Aspiration*
- *Hypertonic saline seems NOT to work*
- *.....and so on and so on*

Things we KNOW that we don't KNOW

- *The right dose of epinephrine in C.A.*
- *The right amount of IV fluids in shock*
- *Which is the best resuscitation fluid?*
- *TXA for bleeding (with a caveat)*
- *TXA for T.B.I.*
- *30:2 vs CCC in OHCA*

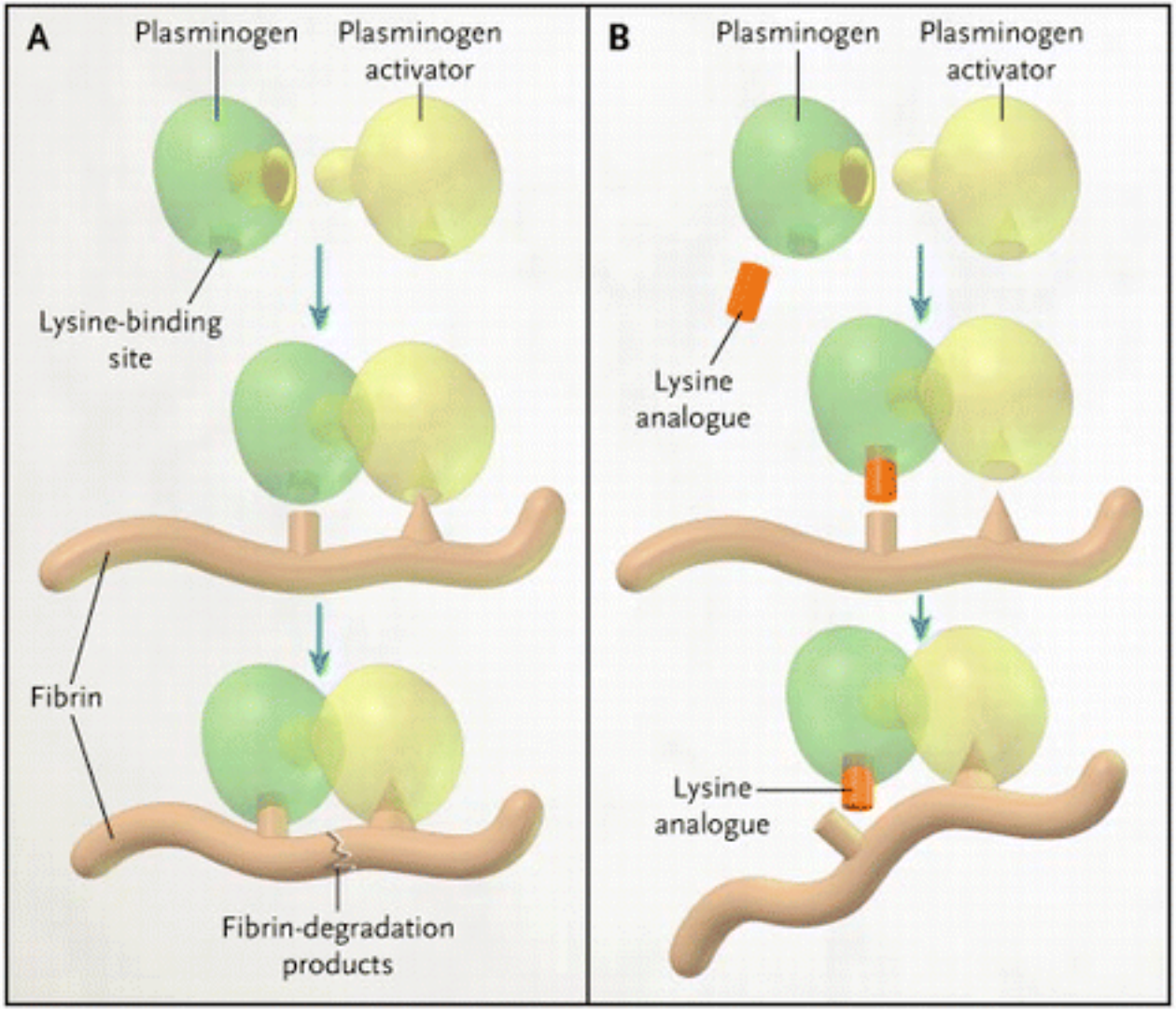
Coagulation Cascade



~ = Fibrinolytic Inhibitors

~ = Fibrinolytic Agents

~ = Coagulation Inhibitors


A
Plasminogen
Plasminogen activator
Lysine-binding site
Fibrin
Fibrin-degradation products
B
Plasminogen
Plasminogen activator
Lysine analogue
Lysine analogue

Things we don't KNOW that we don't KNOW

Is there a role for intermediary metabolism Rx? (Glucose-Insulin-K+ didn't work for STEMI)

Should we monitor the breakdown products of the various organs (TBI, kidneys)?

Is there a future fluid out there that will be the ideal resuscitation fluid?

Etc etc etc….every tissue….every condition

Future Directions

Ideal Resuscitation Fluid

Meddling around with the clotting cascade??

The ideal advanced airway (quick, 100% reliable)

Smart monitors: Central hub of decision-making

Intermediary Metab Support

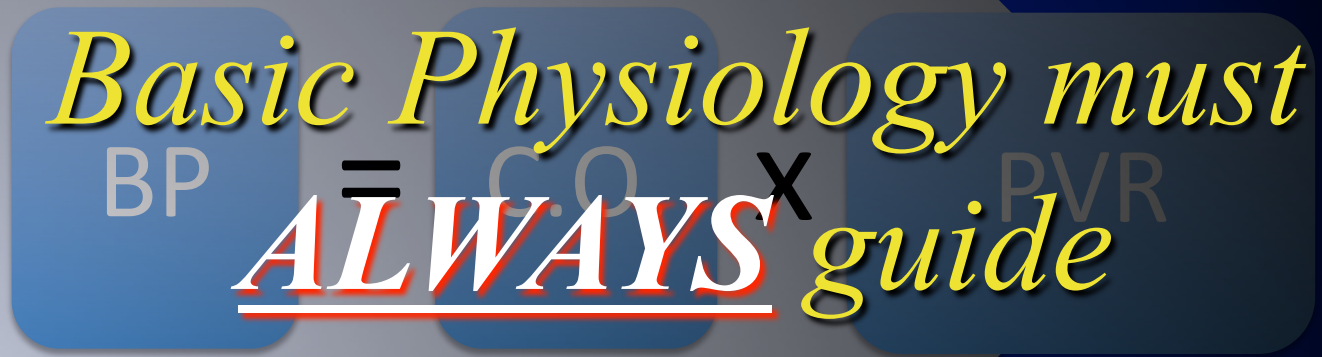
The search for a safe and effective blood substitute

Tissue Oxygenation monitor

Ultrasound in the field!?!?!?

Control apoptotic processes

The Understanding of Basic Physiology must ***ALWAYS*** *guide Evaluation and Management*

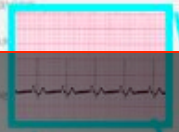


Pace or Cardiovert?

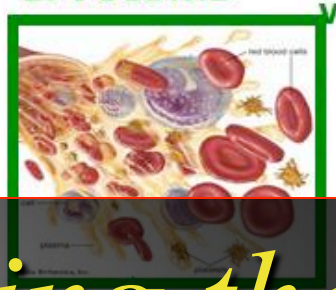
unstable brady (<40)

unstable, non-sinus tachy (>170)

1. HEART RATE



2. VOLUME



U/S for IVC & LV volume

hypovolemia?

hemorrhage? → FAST, AAA?

**IVC >>> LV volume if there is obsxn to flow through heart or lungs …

Tamponade?

New RV infarction?

Massive PE?

Asthmatic alveolar air

Tension pneumothorax?

Can we bring the comprehensive process of patient assessment to the field?

If not now, when?

4. SVR ⬇

** When 1-3 are okay, then volume is going forward … so consider peripheral vasodilation +/- mitochondrial dysfxn

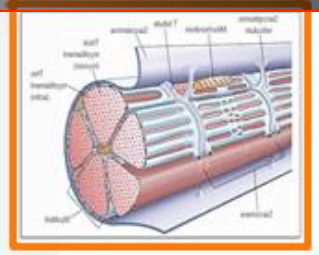
Cervical spinal cord compression?

Anaphylaxis?

Fulminant liver failure?

Sepsis?

Mitochondrial poison (Cyanide, CO)?



3. CONTRACTION

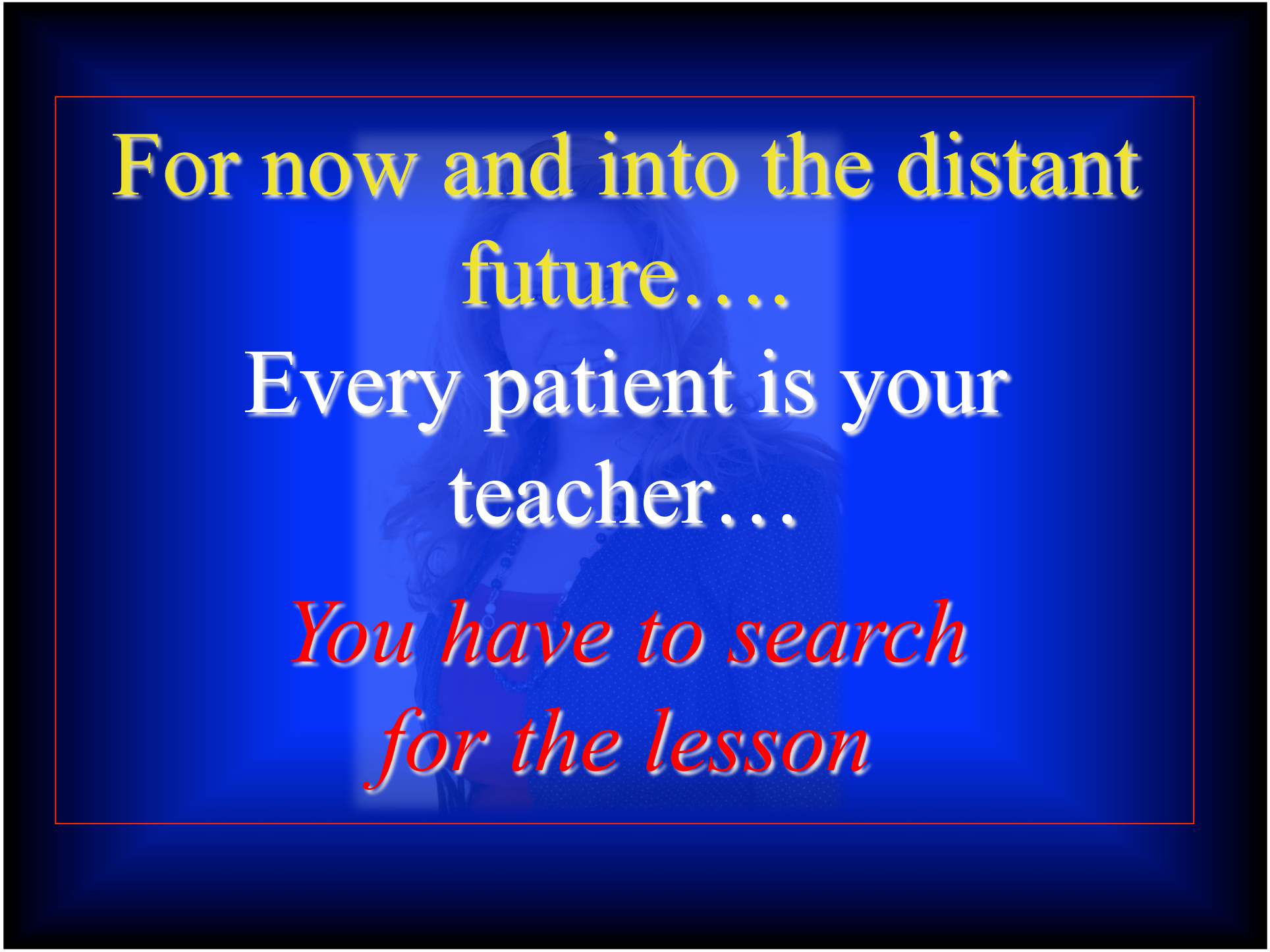
ECG & U/S for LV fxn

Volume in lumen during diastole?

LV walls collapse well in systole?

Is volume truly going forward?

(listen for regurg murmur & rales)


For now and into the distant future….
Every patient is your teacher…
You have to search for the lesson


www.rayfowler.com